THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—28TH YEAR. SYDNEY, SATURDAY, FEBRUARY 1, 1941. No. 5.

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The Halford Oration.[1]

HALFORD THE MAN.

By A. L. KENNY, C.B.E., M.B., Ch.B. (Melbourne), F.R.A.C.S.

WE cannot look, however imperfectly, upon a great man without gaining something by him. He is the living light-fountain which it is good and pleasant to be near. The light which enlightens, which has enlightened, the darkness of the world; and this, not as a kindled lamp only, but rather as a natural luminary shining by the gift of Heaven; a flowing light-fountain, as I say, of native original insight, of manhood and heroic nobleness.

THOMAS CARLYLE.

Explorers beholding a mighty river endeavour to trace it to its source, ascending tributary after tributary to its origin in a lake or in a mountain clad with snow and ice, and in proportion to the mightiness of the river they expect to find more and more tributaries and streamlets coming from a wider and wider area. Occasionally even a great river may be traceable to one small spring—no tributaries or streamlets, no wide area of country. So it is with regard to men and women of genius. Recently, in an oration, I was able to trace antecedents contributory to the great ability of the subject of the oration through both sides of the family to the twelfth century. With the subject of tonight's thirteenth Halford Oration, Professor George Britton Halford, my researches bring me to the single spring; Halford's genius (and he has been acclaimed by most competent judges to have been a great genius) was evolved by himself from his innermost being. I have been unable, after diligent search, to discover any trace of tributary or streamlet of science, and more especially of experimental physiological science, anywhere in his ancestry. His parents and his forbears were the sound, steady, solid men of business, so typical of England for the whole of its history—good, sound, resolute, clear-thinking, "word-is-his-bond" sort of men, with the courage of their opinions and a proper appreciation of their duty to their Creator, to their family and to their country.

Such evolution of genius in individuals whose environment, origin and descent have appeared to be completely devoid of influences competent to lead to this development, are recorded over and over again in the pages of history, more especially in art and in music. From his earliest youth George Britton Halford was attracted by research; he wanted to explore and to ascertain the reason for things; he gave himself up wholly to this attraction.

There is no question as to his genius in experimental physiology—that is, the experimental study of the functions of the living body and of all its parts and constituents. To what heights that genius might have carried him, if he had given full flight to it in the world's capital city, London, untrammelled by the ordinary cares of a family and the responsibilities of the creation and development of a great medical school in a new and strange country, is a most attractive speculation. Had he remained in the world's metropolis, with its vast advantages and the assured reputation he had gained with those most competent to judge him, with his feet already up many of the rungs of the ladder of fame, it is impossible to say to what heights he might have risen in experimental physiology, in which he proved himself to be so very far in advance of his time.

Son of Mr. James Halford, of Haverstock Hill, London, George Britton Halford was born on November 26, 1824, at Petworth, one of those small towns most characteristic of Sussex, with its narrow, winding old-fashioned streets of oak-timbered buildings, grouped prettily around a market

[1] Delivered on November 12, 1940, at the Australian Institute of Anatomy, Canberra.

house built by one of the Earls of Egremont, perched on the side of a low hill at the foot of the great clay-soiled, oak forest-clad weald which separated Sussex from Surrey. The church is large and has a modern spire. The manor houses are built along the southern face of the weald. Petworth House or Manor thus overlooks the little town; though it is large and dull, its splendid park contains the lake which Turner painted. The manor for a long time belonged to the Northumbrian Percies and subsequently came to the Wyndham family, who now own it (the second Lord Leconfield) by way of the Dukes of Somerset. It enshrines one of the most wonderful private collections of pictures in England (more than 600), a fine collection of Reynolds, some Grinling Gibbons carvings, some landscapes by Hobbema, Hogarth's celebrated portrait of Peg Woffington, and four views of the Thames by Turner. Petworth came comparatively late in the history of the development of the country, and was not heard of till the Middle Ages. The country round it, especially to the south down the West Rother and Arun River valleys, is extraordinarily charming. Petworth is west of Stane Street, the only fully known Roman road from Chichester in the south via Pulborough (at the junction of the West Rother and Arun Rivers), where it crosses the Pulborough Bridge, continuing in a north-westerly direction to London; it is a hard, straight Roman road ("a stone" = stan or stan-street). The West Rother, running south of Petworth, turns one of its mills. There is still a definite Roman camp between Petworth branch line and the main Chichester line of southern railway, just before the junction of the rivers I have mentioned. Petworth marble is famous; it is a calcareous stone formed by a deposit of fresh water shells and taking a high polish, and much of it was employed in the building of Canterbury Cathedral, Chichester Cathedral and Petworth House. Just before the Reformation the gentry of Petworth were the Dawtreys, who founded Hardham Priory and whose name proceeds from the high banks (*"d'haute rive"*) of the water meadows of the Arun, which flows south through Arundel to the sea at Littlehampton. Of all the owners of the land in Sussex, the Shelleys have not only glorified Sussex by producing at the end of their line her chief poet, but they have also welded themselves into the soil of the country. Shelley, whose great name might almost add something to the splendour of the country in which he was born at Field Place, will be remembered because that birth of his was next to Horsham. The story of his family will show how widely it was spread over Sussexland, and how worthy it was of inheriting such skies and such a landscape as could produce a master of verse. The name, oddly enough, is from Kent; indeed, there has been, since the centuries after the Conquest, a continual movement westward from Kent into Sussex, of which the Shelleys are but one example. Long before family names arose, while men were still called by their Christian names, and their land was mentioned after them, the men of Shelley in Kent were lords of Shelley. They were there at the end of the thirteenth century; they were there until the middle of the fourteenth century; at that epoch one John Shelley went west for the good of England. The Lancastrian usurpation, that watershed in English social history, is apparent here. John Shelley was returned for the Commons of Rye, just after Agincourt. He had a son who went still further west, and coming to Mitchelgrove, married the daughter and heiress of the lord of that place, a certain John, who took his name, as was right, from his own land. This settlement of the family endured during the Reformation. After this latter date, the Shelleys married into Buckhurst; still further on, before the Civil War, they exchanged their Warwickshire lands for further Sussex holdings; in the eighteenth century one finds them marrying into Maresfield. Already they had a hold upon Findon. Up on St. Leonard's Forest you find their name in one of the first of the ploughed lands that open that deserted belt.

No peasant in the world is so rooted in his customs, and so determined to maintain them, together with the full warm accent of the south century, as is the Sussex peasant. I resist, with great difficulty, the temptation to speak of Rudyard Kipling in these notes of Halford's most interesting as well as beautiful county of origin, Sussex. But I can let you realize what Kipling's beautiful home at Burwash (Burrish) meant to him:

> Each to his choice, and I rejoice
> The lot has fall'n to me
> In a fair ground—in a fair ground,
> Yea, Sussex by the Sea.

In this Sussex and at Petworth, Halford grew up in a natural English way, fond of and excelling in field sports. He was a good cricketer; in Melbourne he used to go to the Melbourne Cricket Ground and put a half-crown on a single wicket for Samuel Cosstick, a famous Melbourne Cricket Club professional round-arm bowler of speed. He was a good sculler and clever at sailing a boat. He could teach youth how to use a fowling piece with safety and skill and well-directed aim. He rode a horse well and was a first-class well-trained whip. Though not a performer, he was very fond of music and would insistently call for a song. If he did not lead in a dance, his eyes followed dancing with the keenest enjoyment. Youth in any form, especially in action, was to him beautiful. All nature appealed to him, and he marvelled at its illimitable aspects. He watched the sunsets intently, looked for the stars as they became visible and took great pains to know them; "A child of nature, he took after his mother". He could hold converse ably on navigation and was able to use a sextant. Of religious mind, he was humble before his God, whom he described as the Almighty Creator; intolerant of religious discussions he would say to his family: "Just have faith, my children." In the whirlpool of the evolutionary controversy he was not Darwinian; he was independent as was his great instructor in comparative anatomy, Professor Owen. To speak of anything before him without knowledge and experience (especially experimental) was to be rebuffed quickly and severely. He disagreed with Mark Twain, who said: "All you need for success in life is ignorance and confidence." A good linguist, especially in the use of the French language, in his younger days he had spent some time at school in Paris. His mother was responsible for much of his early teaching. He was not good at drawing or painting. He began the study of medicine at St. George's Hospital, London, in 1842, at the age of eighteen years. Professor W. A. Osborne, Emeritus Professor of Physiology in the University of Melbourne, delivered the first Halford Oration in Canberra on November 26, 1928, and dealt fully, accurately and exhaustively with the life and work of George Britton Halford, producing all that was known of his medical career, and authoritatively expounding and critically analysing his marvellous original experimental physiological work and discoveries; this may be consulted, by anyone interested, in THE MEDICAL JOURNAL OF AUSTRALIA of January 19, 1929.

The Professor's wife was a very beautiful lady; a splendid large photograph of her, taken in London, is treasured by Dr. G. A. B. Halford, of East Malvern, her eldest son. She had a charming soprano voice of high pitch, with which she was pleased to entertain her family and friends. Her aunt, Madame Anna Bishop, was similarly equipped, and at one time made a professional tour of Australia. Fond of dramatic performances, the Professor constructed a stage and theatre in the backyard of his house, where he trained his daughters and sons and his neighbours' children to act plays, for which he wrote the prologues. He was always human, agreeable and pleasant with his students. I attended his lectures in physiology in 1883 with great interest and profit, and also with much enjoyment, because of his lively sense of humour and his quick wit. Once in each year he unfolded with great gusto and pleasure on the table of the physiology lecture room his much-treasured copy of *The Times*, containing a round-by-round report of the great historic prize fight between Tom Sayers and J. C. Heenan. Much physiology intimate to this fight was discussed and expounded to us, to the mutual delight of professor and students. In 1881 some of the English judges declared attendance at a prize fight to be an illegal act. Just after the fight in 1860 a member of the House of Commons asked the Prime Minister (Lord Palmerston) if it were true that he had contributed to a testimonial raised for

Tom Sayers. Lord Palmerston answered in the affirmative, because he believed in the practice of self-defence; his Home Secretary, Sir George Cornewall Lewis, opposed that view.

Tom Sayers, an Irish Cockney, born at Brighton on July 17, 1826, the great bare-knuckle champion, a wondrous fighter although only a middle weight, had soundly beaten nearly every man who stood up to him, irrespective of height or weight. He fought his last great battle against America's gigantic and skilful young champion, "the Benicia boy". From 7.24 a.m. to 9.45 a.m. they fought, the little man and the big; there was no separating them in merit and the fight was declared a draw. Sayers's admirers at the fight collected and gave him £3,000 on the condition that he never fought again. There was a subscription from Victoria. Tom Sayers was a slogger, rarely weighing more than eleven stone; he leaped into fame when he trounced "the Tipton Slasher" in 1857. He died on November 11, 1865, aged thirty-nine years. Honest, manly, hard-hitting, straight-going and nobly courageous, he was a modest London bricklayer who became one of the world's greatest pugilists. He was beaten only once, by Nat Langham, after 61 terrific rounds. Heenan, although he did fight again, had little success and did not live more than five years after his battle with Sayers.

> Scorning all treacherous feud and deadly strife,
> The dark stiletto and the deadly knife,
> We boast a science sprung from manly pride,
> Linked with true courage and with health allied,
> A noble pastime, void of vain pretence,
> The fine, old English art of self-defence.

Immortal Virgil, glowing with enthusiasm, sat him down to describe a prize-fight. To a crowded Colosseum on great Cæsar's birthday cometh our Poet, acknowledging with dignity his welcome from the assembled crowd, and seats himself there, great as Homer, peer of our unborn Shakespeare, dignified by imperishable achievement. He opens his mouth! He roars! He shouts! He stands, to flourish long arms like those of mere humans about him—to acclaim the entrance of two naked young giants into the sanded arena. These giants raise their arms in salutation—arms girt from the elbow with interwoven straps criss-crossing down to huge fists, there armed with formidable knobs and spikes of brass; they are the celebrated pugilists, the dreadful weapons on their fists the *cæsti*, the gauntlets of death—the boxing gloves of that ferocious age. When the combatants cease and the shouts and screams of the excited multitude die down, behold! One man lies prostrate, limbs asprawl, the trampled sand very red. Our Poet, amid the universal jubilation, sits pensive, head bowed, heedless of all but his thoughts. Truly! for here sits genius, the Seer and Poet, dreaming with prophetic vision of a coming era, of a kindlier world peopled by beings of loftier mind. . . . Hold! See where his neighbour turns to him in reverent question! Hark we to their majestic Roman periods:

> Alas, poor Entellus! Yonder they drag him to a final oblivion. You, Sir, had backed him—heavily, I think?

Answers our Poet, pensive and sighful:

> Sir, in verity! Every available denarius! . . . Confound Entellus!

William Perry ("the Tipton Slasher"), champion of England, was born at Tipton on March 21, 1818. His height was six feet two and a half inches, and his weight fourteen stone six pounds. He died at Wolverhampton on January 18, 1881. Tom Sayers, middle-weight, in height five feet eight and a half inches, well under eleven stone in weight, challenged Perry, to the latter's great amusement. He was so sure of beating Sayers that he sold his public house and everything he possessed to back himself. They fought on a small island at the mouth of the Thames on June 16, 1857. Perry had a K-leg, which bent inward at the knee. The second round lasted for nearly half an hour. For one hour and forty-two minutes they fought, and Sayers became champion of England.

Sayers and Heenan fought their great fight at Farnborough on April 17, 1860. John C. Heenan ("the Benicia Boy", from the Benicia workshops of the Pacific Mail Steamship Company) was born in West Troy, United States of America, in 1834. He stood six feet two inches and weighed thirteen stone seven pounds. He married Adah Isaacs Menken, the celebrated beauty, who was the Mazeppa at Astley's Circus, and was a friend of Charles Dickens and of Algernon Swinburne; her name was associated with the mad King Louis of Bavaria. Heenan died on October 28, 1873, at Green River Station, Wyoming, United States of America.

People packed into a special train of 33 carriages at London Bridge Station at 4 o'clock on a cold April morning to behold an exhibition of such indomitable courage, skill and endurance, such heroic fortitude as shall live in men's memories, shall be told of and written about for many a long year to come—yea! verily! year after year in the physiology lecture room of the Medical School of the University of Melbourne by the Dean of the Faculty of Medicine, the greatest experimental physiologist of his day—and there were 12,000 present. Sayers lost the toss and had to face his gigantic opponent with the sun in his eyes. Time allows an account of the historic seventh round. One of Heenan's eyes is closing and he drips blood from cut cheek and split eyebrow—his great chest shows the vivid marks of the English champion's powerful fists; Sayers bleeds from an eyebrow and has been knocked down four times. He is now comfortably set, his blows are better timed and harder; he gives Heenan a crushing blow on the nose that nearly lifts him off his legs, and again another flush hit that rocks six feet two inches and sends him reeling. Heenan, recovering, drives a smashing return blow, but Sayers expects it, leaps back, bounds in again; and then out shoots Heenan's terrific right, backed by arm, shoulder and mighty body; Sayers stops it with his right forearm and is instantly numb to the shoulder, his arm swollen and purple. The fight rages ever fiercer; they clinch, and five feet eight inches drives in damaging body blows ere, wrestling, he goes down beneath. The eighth round; Sayers is as quick as ever; but look! he does not use his trusty right; he keeps it across his chest inactive. Round after round Sayers comes up smiling, his right arm across his chest, relying for defence on nimble feet and for attack on his left; his right is done for. His left smashes in, Heenan spins, totters and falls. Next round lasts fifteen terrible minutes; Sayers's arm hangs or dangles limp and cumbersome at his side. Round succeeds round, Sayers down and up again, playing with his solitary fist for Heenan's eyes, slowly but surely blinding him. Sayers is greatly battered; nose and mouth seem one; cool, watching every chance, yet time and again smitten to the trampled grass. Twenty-first round, Heenan is fast going blind. Sayers is weakening fast, yet he rallies and Heenan reels back from the unerring punishing left. Heenan, hands puffed up like boxing gloves, recovers again, but is too weak to do much damage, and Tom, fighting grimly as ever, has Heenan blind at last. Cheers for Tom Sayers, the unconquerable champion still! But that disturbance! Heaven and earth! the police! Off we go! The fight is ended anyhow. What, a draw? Well, hurrah for our Tom, another for the game lad from Benicia: Americans are the right breed after all . . . The physiology lecture room rocks with cheers for the dear old Professor with the dundreary whiskers, worn all round the ring at the fight—the fine old English gentleman of the fine old English sporting school. The radius, the small bone of Sayers's right arm, was broken.

I strongly suspect that our genial Professor was at Farnborough that cold April morning, and that that is why he so carefully preserved *The Times* of the day after the fight, April 18, 1860; its report is acknowledged by all sporting authorities as the most complete one. The Prime Minister of Great Britain, Lord Palmerston ("Pam"), was there with a large number of leaders of the aristocracy—why not our Professor Halford also? Homer did not disdain to describe fights at the funerary gamès, neither did Virgil in the fifth book of the Aeneid; why should I, in Canberra?

In *Punch* of April 28, 1860, there appeared a most amusing, skilful parody of one of Lord Macaulay's "Lays of Ancient Rome"—"How Horatius Kept the Bridge in the Brave Days of Old"—under the title of "The Combat of Sayerius and Heenanus", a "Lay of Ancient London", supposed to be told by a grandfather to his grandson on April 17, 1920; it was ascribed to William Makepeace Thackeray, and later H. Cholmondeley Pennell's name was suggested.

Occasionally, after calling the roll, the Professor would look around the class silently, thinking, and then would

suddenly say: "Well, gentlemen! Suppose we have questions today. You, no doubt, have difficulties in studying your physiology. Come now, ask me any questions on matters that are troubling you, and I will try to explain them if I can." On the one morning that is in my mind at the moment, there was no response to this request for several minutes. The Professor expectantly surveyed the class, but no question came. Taking up the roll book and looking at the last name marked present, he said: "Mr. X has answered the roll call, has he no difficulties in his physiology? What do you say, Mr. X?" "Mr. X", thus singled out from the whole class, became self-conscious and nervous, but blurted out, in a downright manner natural to him, a question of the very greatest difficulty. The class, realizing the possibility of an interesting situation, became acutely alert. Professor thought hard for a minute or two, looked fixedly at "Mr. X", then passed his eyes along the obviously interested members of the class, looked back at "Mr. X", and further upset his mental equilibrium by shooting at him the totally irrelevant question: "Mr. X, *do* you ever read your Bible?" Surprisedly and stammeringly "Mr. X" answered: "Yes, Sir!" The Professor, half turning in his chair, with a very sagacious nod of his head, said: "Then, Mr. X, you will remember to have read that God helps those who help themselves! Next question please."

One of my predecessors in the delivery of this oration, Professor Thomas Cherry, very happily suggested to me some lines of Browning's, of apt application to some of the things I have told you of the Professor. They occur in the second stanza of that delightfully joyful "A Likeness":

> . . . mid the other spoils
> Of youth—masks, gloves and foils,
> And pipe-sticks, rose, cherry-tree, jasmine,
> And the long whip, the tandem lasher,
> And the cast from a fist (not alas! not mine,
> But my master's, the Tipton Slasher),
> And the cards where pistol-balls mark ace,
> And a satin shoe used for a cigar-case,
> And the chamois-horns (shot in the Chablais),
> And prints—Rarey drumming on Cruiser,
> And Sayers, our champion, the bruiser.

In the years from 1886 to 1888 the University of Melbourne received as professors in scientific subjects three very able, remarkable men, in the best years of their lives, who were partially concerned in the training of medical students, who would have added lustre to the staff of any university in the world, and who did imperishably fine work for the University of Melbourne; they increased its fame and received, whilst they were still working for it, the Fellowship of the Royal Society of Great Britain. These were each knighted by His Majesty the King. They were Professor Sir Thomas Lyle (natural philosophy—1888), still happily with us, and Professor Sir Baldwin Spencer (biology—March, 1887) and Professor Sir Orme Masson (chemistry—October, 1886), who have gone to their reward.

One of these related that when he attended his first meeting of the Faculty of Medicine at the medical school, toward the close of the business, some of the members took their leave and hurried away. He, being a stranger, waited for the seniors to leave the room first; they were the Vice-Chancellor (Sir Anthony Colling Brownless), Professor Sir Frederick McCoy, Professor Halford (Dean of the Faculty), Sir Harry Allen and some others. As he was leaving the room immediately behind them, they invited him to follow them as they crossed the road to a hotel well known to University men. They entered a passage, and the senior tapped a small wooden panel, which promptly slid back to show an attentive fair-haired lady who greeted them politely. "Six whiskies", said the senior to the lady, and then turned to my informant: "You will have whisky, won't you?" My friend, taking all this as the Australian form of courtesy to a newcomer, graciously assented, and they drank his health. The panel had been closed; the second senior knocked on it and repeated the order. So it continued until each in turn had given an order. My friend went home ruminating very joyfully upon the characteristics of the environment of a new professor in Australia. *"O tempora! O mores!"*

Professor Halford named his first son George Arthur Billings Halford in affectionate memory of a dear friend, Dr. Arthur Billings of London, a physician of high reputation who had written "The Principles of Medicine", a book on the practice of medicine based on reason; he also wrote a most interesting book on gems and precious stones. Quite near Professor Halford's birthplace, between Petworth and Horsham, is Billinghurst. It is stated that this name may come from a pre-Roman source, like Billingsgate—the worship of a native god, Belinus. But the "hurst", like all the other "hursts" up and down the south of England, and like "ham", is probably Teutonic.

In his early medical days, before there was any mention of his coming to Australia, Professor Halford went as a ship's surgeon to India, and there came for the first time into close touch with snakes and their venom, to which he applied his experimental physiological mind afterwards in Australia. The cobra was the venomous snake most spoken of in India, and in later years the Frenchman Calmette evolved from the venom an anti-venene which cured persons suffering from cobra poisoning. It was found to be inactive against the venom of the krait (Russell's viper), which was usually three or four feet in length, had a larger bite than the cobra, and injected a larger amount of venom of great lethal power and very destructive to the parts bitten. In 1906 I witnessed experiments at the Pasteur Institute at Kasauli on a hilltop opposite Simla in India. At that time it had not been possible to dilute the krait venom sufficiently to make it possible to inject it into a horse without very serious destruction of the tissues at the site of injection. I have seen the shoulder joint of a horse completely exposed by the sloughing away of all the soft tissues of the shoulder covering. There is now an effective anti-krait venene.

India has some magnificent highways, known as grand trunk roads, some running mainly north and south, others east and west, especially from Calcutta to Bombay or from Calcutta to Peshawur in the extreme north-west; they cover 1,000 to 1,200 miles, and have well made, well formed and carefully graded surfaces, with three rows of trees on each side, the branches interlacing over the road, giving much needed and most grateful protection from the intensely hot sun. Innumerable natives of all races, tribes and religions throng these roads all day long, walking from place to place, barefooted and barelegged. No native can be induced for love or money to walk those roads at night for fear of the krait. On moonlight nights it is especially dreaded; the overhead foliage pierced by the moonlight gives a pattern of tracery on the road, the very deep shadow of the branches contrasted with the brightly lighted spaces between. By a most dangerous instinct the krait lies along the shadow and cannot be seen; a native would be at its mercy.

In my days at the medical school at Melbourne the central quadrangle was frequently filled by a motley crowd of small boys and dogs of all breeds that they had captured and brought to sell for the experiments in the investigation of snake venom and its remedy. Some of these investigations were also carried out in the Melbourne jail. In Professor Halford's day the intravenous injection of ammonia, introduced by him, was the means of saving lives. He also used calcium chloride according to an early method of Calmette. Since those days very great advances in the knowledge of physiology and of biochemistry all the world over have made possible equally great developments in research into this problem by Sir Charles Martin, F.R.S., and latest of all by Dr. Charles Kellaway, F.R.S., Director of the Walter and Eliza Hall Institute at the Royal Melbourne Hospital, whose work has placed in the hands of every legally qualified medical practitioner a most reliable remedy for each variety of most of the Australian snake venoms.

A good story is told of a very interesting experience with a snake on the banks of the Goulburn River near Seymour, a place much resorted to for perch and Murray cod, though equally resorted to by venomous snakes. Two young fellows had been fishing for some time unsuccessfully, and they made up their minds to try to get a frog or two for bait, but could not easily find any. Whilst still searching, one of them heard a rustle near a grass tussock and saw a snake

with a frog in its mouth. The snake had got about half the frog into its gullet, but was having difficulty with the remainder. The two young men rapidly sought forked sticks of some length and were soon successful. One man, with the longest stick, pinned the snake, a large tiger, to the ground with the fork at the neck just behind the head, whilst the other, with his forked stick, levered the frog out of the snake's mouth. He told the holder of the snake to keep it pinned, because, snake or no snake, common ordinary decency of behaviour demanded that some recompense should be made to the snake for this forcible stealing of its property. By dint of clever manœuvring they forced the snake's head into an upright position with its mouth widely opened, and from a safe distance one of them poured some whisky from his flask well into the snake's throat and, with a fling, let it go. *Blitzkrieg* from Hitler was as nothing to the speed of the snake in its departure. The stolen bait was successful and there was joy for the fishers. After a good catch or two they ceased fishing to light their pipes, when one of them again heard a rustle. On investigation they found a snake again, as much like the first one as possible, with a frog in its mouth. It is wonderful what a little whisky will make a snake do.

In his examination of the blood of persons bitten by Australian venomous snakes, Professor Halford recorded certain changes which he considered resembled those to be found in certain fatal diseases; this was a momentous observation, the full importance of which was not, and could not, be appreciated until great advances had been made in biochemistry and in the development of the then unknown science of bacteriology. It was then found that these changes were intelligible by the discovery of bacterial toxins, not the bacteria, as a source of disease. This affords one of several proofs of the statement that Professor Halford's genius was far ahead of his time and its methods.

The Professor visited England only once from the date of his arrival to his death; he was granted leave of absence to visit Europe at the end of 1880, and returned in October, 1881. He died in 1910.

The University of Melbourne was inaugurated on April 13, 1855. In June of that year Dr. Anthony Colling Brownless was appointed a member of the Council of the University; he arrived at Melbourne in 1852. He was a Member of the Royal College of Surgeons of England (1841) and a Licentiate of the Society of Apothecaries of London of the same year, and held the degree of M.D. of Saint Andrews (1846), to which was added the degree of M.D. *ad eundem gradum* of Melbourne in 1856. He had been a student and friend of Sir James Paget at Saint Bartholomew's Hospital, a professor and lecturer in anatomy, physiology and pathology in London. Brownless at once started an agitation for the formation of a medical school. It happened that several medical graduates who were very active and prominent in public life in Victoria were also members of the council of the university at this period; they supported Brownless, as also did his colleagues in medical practice. Professor Osborne has detailed the difficulties and disappointments they experienced until in 1861 the project was adopted by the Government. Brownless became Vice-Chancellor of the University on May 31, 1858, and on April 4, 1887, he became Chancellor, holding the position until his death on December 3, 1897.

Sir Anthony Brownless, aided by Professor Sir Frederick McCoy, was responsible for the choice of a five years' course of studies for the degree of bachelor of medicine in the University of Melbourne. On his appointment as professor in charge of the medical school, Professor Halford enthusiastically accepted the five years' course, which was the first of its length in English-speaking countries. Until 1894 the General Council of Medical Education and Registration of the United Kingdom was satisfied with four years; it then specified a five years' course. The medical school of the University of Melbourne persisted with its five years' course until 1932, when it extended it to five years and two terms in a sixth year. This length of course resulted in a high appreciation of Melbourne medical graduates in English-speaking countries. It was included in the act governing the registration of medical practitioners in Victoria. In 1890 Professor Sir Harry Allen, on a visit to England, placed before the General Medical Council a synopsis of the curriculum and course in Melbourne. The president in his address at the next session of the council said (and I would have this kept in mind with regard to recent utterances of the council):

> A really excellent mode of utilizing a five years' period, with its associated periodical tests, will be found in an interesting communication describing the curriculum and examinations now enforced by the University of Melbourne for its degrees in medicine and surgery. This document, which has found place in the minutes of the Executive Committee, might with advantage be sent to the several qualifying bodies and might also be distributed amongst both teachers and examiners.

As a result, the General Medical Council agreed to register medical graduates of the University of Melbourne, provided they were already registered by the Medical Board of Victoria, and it was arranged that the Medical Board of Victoria should register medical practitioners who had been registered by the General Medical Council of the United Kingdom. This was a most valuable and a very highly appreciated boon for all Victorian medical graduates going to the United Kingdom. Registration there gave them the right to practise and to hold hospital appointments. Men wishing to do post-graduate work in Great Britain freely availed themselves of this very great privilege which, whilst allowing them to follow the work of the most eminent surgeons and physicians in the hospitals, made it possible to maintain themselves whilst so doing. The various electoral boards advising the committees of the Melbourne metropolitan hospitals are constantly presented with the written opinions of the most prominent hospital surgeons and physicians of London with regard to the outstanding ability of the young Australian graduates who have acted as resident medical officers of their hospitals.

The General Council of Medical Education and Registration of the United Kingdom enjoys the unique position of being a subcommittee of the Privy Council and has great authority and power. Part II of the act of 1886 was applied to the State of Victoria with effect from April 1, 1890, by an Order in Council made in pursuance of Section 17 of the act on March 21, 1890. Degrees granted in the Faculty of Medicine of the University of Melbourne were recognized by the General Council of Medical Education and Registration of the United Kingdom under Section 13 of the act on May 26, 1890. This General Council controls 27 licensing bodies (18 universities and nine medical corporations) in the United Kingdom of Great Britain and Northern Ireland and in Eire.

The General Council of Medical Education and Registration, *inter alia*, has power to alter the standards of medical education it had ordained for those nine licensing bodies or medical corporations, and it has used that power. When Herr Hitler and the Nazi party expelled Jews from Germany and other countries under their control, a number of medical practitioners with diplomas of varied merit and of different degrees of professional skill sought to practise their profession in the United Kingdom and in the territories of the British Commonwealth of Nations. They applied for registration in Great Britain; some were registered; others whose degrees or qualifications did not satisfy the requirements of the General Medical Council endeavoured to obtain British degrees at universities or diplomas from medical corporations. The medical profession in Great Britain, alarmed at the influx and the consequent interference with their means of livelihood, made their alarm felt in Parliament and in the General Medical Council. The Home Secretary limited the number of foreigners (not British subjects) allowed to enter Great Britain and later prohibited their entry. For a time a strictly limited number had been permitted to remain for fourteen months in Great Britain preparing for and passing the necessary examinations for the lowered standard approved by the General Medical Council for the "triple qualification" (Licentiate of the Royal College of Surgeons and of the Royal College of Physicians of Edinburgh, and of the Royal Faculty of Physicians and Surgeons of Glasgow). On receipt of these diplomas they were registered for practice in the United Kingdom by the General Medical Council, but were not allowed to remain to practise in the United Kingdom.

They were told that this registration would secure registration for them in the dominions, and they came to Melbourne and elsewhere in numbers. In the view of the members of the Medical Board of Victoria such men were not eligible, under the *Medical Act*, to be registered in Victoria. One of them appealed against this decision to a Supreme Court judge in Victoria, who upheld his appeal and ordered his registration. The appellant's lawyer and the judge both expressed their strong opinion that the act urgently needed amendment. The Medical Board of Victoria applied to the High Court of Australia for leave to appeal against the Victorian Supreme Court judge's decision, and leave was granted. The six judges of the High Court divided evenly on the appeal, three to three. Therefore the decision of the Supreme Court of Victoria stood. In these circumstances the Parliament of Victoria amended the *Medical Act*, and this amended act has been in force since 1938.

The General Council of Medical Education and Registration has objected to this act of 1938 as being a breach of the arrangement for reciprocity made in 1890. It certainly prevents the registration, without five years' regular course of study, of medical practitioners registered for the "triple qualification" by the General Medical Council, who are not allowed, despite being so registered, to practise in the United Kingdom, but whom the General Medical Council insists are entitled by agreement to registration in Victoria.

Thus the prestige attached to the high standard of a five and two-thirds years' course of studies is in danger of being lowered. This state of affairs implies having two standards of medical registration in Victoria. Discussion on the matter is in abeyance until after the war has ended.

I conclude with the words of Tacitus (A.D. 52 to 117):

To bequeath to posterity a record of the deeds and characters of distinguished men is an ancient practice which even the present age, careless as it is of its own sons, has not abandoned.

I have endeavoured to aid the sons of Professor George Britton Halford in this ancient practice by this attempt to sketch and suggest his character.

LOCAL IMPLANTATION OF SULPHANILAMIDE FOR THE PREVENTION AND TREATMENT OF GAS GANGRENE IN HEAVILY CONTAMINATED WOUNDS: A SUGGESTED TREATMENT FOR WAR WOUNDS.

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AND

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Adelaide.

SINCE the outbreak of war the treatment of large and heavily contaminated wounds has once again become a matter of great importance, and the prevention of infection in such wounds is still largely an unsolved problem. This paper is the report of a series of experiments designed to determine the value of local applications of sulphanilamide for this purpose. Throughout the experiments treatment has been applied in a way which would be practicable in war.

The experiments were carried out at Woodside Camp, South Australia, in the authors' leisure time during training with a field ambulance of the Australian Imperial Force. These circumstances have made minor deficiencies unavoidable. For instance, the number of animals used in each experiment had to be related more to the working time available than to the real requirements of the experiment. The laboratory in which the work was done was situated in a tent, and there was no means of protecting the animals against the very variable weather, which probably contributed to the deaths of several from post-operative pneumonia. However, significant results were obtained in most of the series. The method of treatment adopted probably constitutes the most effective reply to the present urgent problem of wound infection, and this justifies the publication of the results in their present state.

[1] Aided by a grant from the David Murray Scholarship Fund of the University of Adelaide.

Historical.

During the last few years a great deal of research into the effects of oral and parenteral administration of the sulphonamide group of drugs has been carried out, and it is remarkable that such scant attention has been paid to the effect of the sulphonamides applied locally.

From time to time articles have appeared describing the local use of the drugs in the treatment of various infective conditions, but Jensen, Johnsrud and Nelson[1] were the first to use sulphanilamide prophylactically in wounds. They treated a series of thirty-nine compound fractures by excision of the wounds followed by local implantation in each case of 10 to 20 grammes of powdered crystalline sulphanilamide. In this series there were no cases of primary infection, although two wounds did become infected secondarily. For the previous ten years the incidence of primary infection in the Minneapolis General Hospital, where this treatment was carried out, had been about 25%. Jensen followed up these clinical findings with experimental work which will be considered in detail later.

This remained an isolated report until the outbreak of the present war made wound infection a subject of urgent importance. The French, who were stimulated first by their proximity to the fighting, carried out several investigations. Levaditi and his collaborators[2] studied the effects of local treatment with large amounts of rubiazol and sulphapyridine in war wounds already infected with streptococci and other organisms. They describe their results as "particularly satisfactory", but found that sterilization of the wound was not always achieved, as chemo-resistant strains of organisms existed.

Nitti,[3] using rabbits as laboratory animals, investigated the effects of sulphanilamide applied locally to both clean and infected wounds. He found, in the case of wounds inoculated with hæmolytic streptococci, that the infection was either greatly delayed or prevented.

Some time after our experiments were begun, Colebrook[4] published a letter in *The Lancet* summarizing the work of Legroux,[5] who has carried out extensive experimental work on this problem, and applied the results to the treatment of war casualties. Contused wounds in laboratory animals were infected with cultures of hæmolytic streptococci and of *Clostridium welchii*, and were treated by dusting with sulphanilamide powder. With the hæmolytic streptococci it was found that animals treated with a single dusting at the time of infection died thirty-six to forty-eight hours later than the controls. Further local treatment, combined with the administration of sulphanilamide by mouth, saved the animals' lives. Similar results were obtained with infections by *Clostridium welchii*. A considerable number of French wounded were treated along these lines during May, 1940. No figures are available, but the results are said to have been very encouraging.

At the Mayo Clinic[6][7] during the last year sulphonamides in solution or suspension have been used locally in the treatment of infected wounds. The results were not uniform; in most cases reported there was a sudden and striking improvement. Promising results were also obtained when sulphonamides were used prophylactically after operations in which post-operative suppuration was expected.

Properties of Sulphanilamide.

Sulphanilamide is a white crystalline powder soluble in water to the extent of 0·8% at body temperature. The pH of a 0·5% aqueous solution is 5·8 to 6·1. *In vitro* it will inhibit the growth of many pathogenic organisms, being especially effective against certain hæmolytic streptococci. Fildes's[8] work suggests that it acts by interfering with an essential metabolite, probably *p*-aminobenzoic acid, and thus inhibits bacterial growth.

The concentration of sulphanilamide in solution is an important factor in its effect on bacteria, and as higher

concentrations are used a greater variety of organisms is affected and their growth is more strongly inhibited. This is well illustrated by Spray's[8] work on the effect of sulphanilamide on the growth of anaerobes *in vitro*. He found that at a concentration of 0·4% the growth of all the pathogenic anaerobes was inhibited if not entirely prevented. As progressively lower concentrations of the drug were employed, one after another of the organisms was able to grow. In this connexion it is worth noting that the local use of the powder will give a concentration in the wound exudate of more than 0·5% (Jensen, Johnsrud and Nelson[1]).

Effects of Application of Solid Sulphanilamide to Open Wounds.

It has been shown by Nitti[3] that solid sulphanilamide applied directly to wounds does not cause any local irritation. Wolff and Julius[10] found the growth of cells in tissue culture to be unaffected by the drug in a concentration of 1:1,000, although the growth of streptococci in blood was inhibited by a 1:50,000 solution. These authors also demonstrated that sulphanilamide in a concentration of 1:500 had no effect on phagocytosis. These results are all in contrast with the effects of the commonly used antiseptics.

Russell and Falconer[11] have shown that the local application of solid sulphanilamide to the rabbit's brain does not cause any appreciable damage to the tissues, although the prolonged presence of solid particles may give rise to some foreign body reaction.

In experimental compound fractures in rabbits and dogs, Key and Burford[12] found that the local implantation of sulphanilamide did not interfere with healing and lessened considerably the danger of infection.

Clinically, Jensen, Johnsrud and Nelson[1] noted no adverse effects following the use of large amounts of solid sulphanilamide locally in compound fractures, and the wounds they treated by packing with the drug healed by first intention without signs of inflammatory disturbance. The local instillation of a saturated solution of sulphanilamide into the thoracic cavity after pneumonectomy, according to Harrell and Brown,[7] had no deleterious effects.

In order to satisfy ourselves of the harmless nature of the drug and particularly of the product we were using, the following experiments were performed.

For the first experiment three guinea-pigs were used, and in each an incision about two centimetres long was made just to the left of, and parallel to, the spines of the middle thoracic vertebræ. The incisions were carried deeply into the *erector spinæ* muscle. Each wound was packed with 0·5 gramme of powdered sulphanilamide and the skin was closed with silk thread. In each case a similar incision was made on the right side and closed without the insertion of sulphanilamide. One animal was killed on the fourth day and the whole region of the wounds was removed in a block and examined microscopically by Dr. J. B. Thiersch. His report indicated mild inflammatory changes in the region of both wounds, but the changes were less extensive and less pronounced in the wound that was packed with the drug. The wounds of the two remaining animals were reopened and examined on the eighth and twelfth days. No difference could be detected between the treated and the control wounds either in the appearance of the healing surfaces or in the strength of the scar. Twenty-eight days later all wounds were perfectly healed with fine scars.

In a second series of four animals a small right paramedian laparotomy wound was made and 0·5 gramme of powdered sulphanilamide was packed into the peritoneal cavity and left lying against the anterior abdominal wall in the left inguinal region. The animals were sacrificed one, three, five and ten days after operation. In each case there was some localized inflammatory change with adherent omentum or intestine in the immediate vicinity of the peritoneal wound. In all other respects the peritoneum of every animal was quite normal, and in the left inguinal region, where the bulk of the drug had been packed, there was no sign of inflammatory change. In the animal examined twenty-four hours after application of the drug, no trace of the powder could be seen.

In order to see whether it had any local stinging effect, powdered sulphanilamide has been placed in a number of open wounds and abrasions at the casualty department at the Royal Adelaide Hospital. The applications were quite painless and were not followed by any adverse effects.

These findings are in complete agreement with those reported by other authors, and it may be concluded that powdered sulphanilamide is non-irritating to wound surfaces and to body tissues generally, does not cause any detectable inflammatory reaction, and does not interfere with wound healing.

Rate of Absorption of Sulphanilamide Applied Locally.

The blood concentrations of sulphanilamide were determined by Jensen, Johnsrud and Nelson[1] in two cases after ten and six grammes of sulphanilamide had been buried in operative wounds. They found that the highest blood level was reached in about eighteen hours and thereafter the blood sulphanilamide fell slowly and the drug could still be detected in the blood at the end of sixty hours.

Nitti,[3] working with rabbits, obtained similar results. He found that a steady rise in the blood sulphanilamide level, reaching a maximum concentration in about twelve hours, was followed by a slow progressive fall, and the sulphanilamide was not completely eliminated until forty-eight to sixty hours had elapsed. These results are in contrast with the well known rapid rise and fall in blood sulphanilamide level following oral administration.

In the series of experiments reported here the blood sulphanilamide content was not determined; but it was noted in biopsy or at post-mortem examination of wounds which had been packed with the drug, that a large amount of sulphanilamide was present at the end of twelve hours, that a good deal was still present at the end of twenty-four hours, and that in some large wounds which had been packed with one gramme of sulphanilamide the powder was still visible at the end of four days. Thus for several days solid sulphanilamide is present in the wound; it follows that the wound exudate during that time must contain an almost saturated solution. From this local depot of sulphanilamide there is a slow and prolonged absorption into the blood.

Nitti[3] also found that the toxicity of sulphanilamide implanted locally in rabbits was considerably lower than that of sulphanilamide administered orally and was in the vicinity of five to six grammes per kilogram of body weight.

Effects of Sulphanilamide in Infected Wounds.

The harmlessness of solid sulphanilamide to the tissues of the animal body having been established, it remains to determine the effect of its local application in controlling wound infections. This has been done in a series of experiments described below, in which gas gangrene, staphylococcal, streptococcal and mixed infections are in turn considered. The results are discussed and compared with those of other authors using local and oral therapy.

Technique.

Guinea-pigs were used as laboratory animals. The operations were conducted under ether anæsthesia without premedication. An attempt was made to imitate the type of wound which occurs in war. A piece of detached crushed muscle was left loose in every wound, the surrounding tissues were severely traumatized, and foreign bodies in the form of earth were rubbed into the damaged tissue. In many of the animals a comminuted fracture of bone was produced as well; but owing to the high incidence of post-operative pneumonia, it was considered essential to shorten operative time as much as possible so that bone injury was avoided where a subsequent excision operation had to be performed.

Gas gangrene infections occurring naturally are caused by spores of the anaerobes. Attempts to use spores in these experimental wounds were not successful, and we therefore used virulent cultures of the organisms in order to be certain of producing an infection in every case. This is, of course, a far more serious infection than could ever occur naturally, for not only is there no delay period for

germination, but the wound is damaged at once by preformed toxin in the culture medium.

An interval of twelve to fourteen hours was allowed to elapse before excision in all experiments in which this operation was performed. This corresponds to a longer period in naturally occurring wounds, in which some six hours elapse before many organisms can be found in a direct smear. This time interval was chosen in order to conform to the policy of applying only such treatment as would be practicable in war, for under active service conditions it should be possible to transport most wounded men to an operating centre within twelve to eighteen hours of the time of injury.

The excision in cases in which no extension of the infection was found, was such as would be normally performed upon an accidental wound—that is, all soiled and traumatized tissue was removed. When extension of the infection had taken place, as in the animals infected with *Clostridium œdematiens*, as much as possible of the diseased tissue was cut away. Primary closure of all wounds was attempted after excision.

Details of the source of the organism and of the operative technique in the different series of experiments are set out in the various sections which follow.

Death was taken as the end point in the experiments, and the time of death was carefully noted in all cases. Post-mortem examinations were carried out as soon as possible, particular attention being paid to the condition of the wound, the muscles of the leg and abdominal wall, and the viscera. Smears were taken from the wound, the subcutaneous tissue in the groin, the peritoneal fluid, the peritoneal surface of the liver, and other tissues showing evidence of pathological change. The heart blood was examined and attempts at culture from it were made in most cases.

Gas gangrene was incriminated as the cause of death when findings comparable with those described by Topley and Wilson[12] were noted. These cases are labelled "G" in all graphs.

Of the animals on which excision of the wound was performed, a large proportion died of pneumonia. All of these animals, labelled "P" on the graphs, had shown respiratory symptoms before death, and post-mortem examination revealed no extension of the organisms or pathological effects of gas gangrene. In addition, in all these cases a gross patchy consolidation occupied the greater portion of both lungs. When lung consolidation and signs of gas gangrene were coexistent, the cause of death has been given as gas gangrene.

These pneumonic deaths have made statistical analysis of the results difficult, and this difficulty could be overcome only by a repetition of the experiments, which time did not permit. However, it was decided that the greater length of life of these animals compared with the controls, and the complete absence in them of post-mortem signs of gas gangrene, justified the assumption that, for statistical purposes, the animals which died of pneumonia could be considered as having survived the experimental infection with gas gangrene organisms. The short statistical analyses which follow each section have been worked out on this assumption.

More reliance can be placed on the findings in the small *Clostridium welchii* and *Clostridium septique* series, because of the highly significant result obtained later in the larger series of experiments with a mixture containing both these organisms.

Clostridium welchii.

The *Clostridium welchii* experiments were carried out as follows.

In the first series of experiments a suspension of spores containing approximately 2,000 million spores per cubic centimetre was used. This suspension was prepared from stock culture "L2" from the collection of the Institute of Medical and Veterinary Science, Adelaide—a culture isolated from a fatal human case of gas gangrene in 1939. Difficulty was experienced in securing constant infection with spores. Only two out of 30 animals in which spores were added to a wound containing sterile earth, loose crushed muscle and broken bone, contracted gas gangrene, in spite of the fact that calcium chloride was added to some of these wounds.

A virulent culture of bacilli was therefore used. An animal was killed by the intramuscular injection of 0·1 cubic centimetre of the spore suspension and 0·1 cubic centimetre of saturated calcium chloride solution. Its heart blood was incubated in cooked meat medium for twenty-four hours at 37° C., and two drops of this culture were used. Sterile garden earth was obtained by the autoclaving of a sieved specimen for three-quarters of an hour at fifteen pounds' pressure.

An approximately transverse incision about two centimetres long was made over the left hindquarters just behind the crest of the ilium. The incision was carried through the skin and subcutaneous tissue deep into the muscle. The gluteal muscle mass was cut across and a piece of the muscle was removed, crushed, and replaced in the wound. The remaining muscle exposed in the wound was crushed with a heavy artery forceps. As much sterile earth as could be carried on a short tenotome blade was placed in the wound and rubbed into the tissues. Two drops of the broth culture of bacteria were then added, and the wound was temporarily closed by forceps and forcibly massaged for a few moments. In the control group the skin wound was then closed with Michel clips. In the treated animals 0·5 gramme of powdered sulphanilamide was packed into the wound before final closure.

In five of the animals (numbers 69 to 73) which had been treated with sulphanilamide at the time of the first operation, excision was performed twelve hours later. Skin edges were cut away and the subcutaneous and muscle tissue was excised until the muscle appeared normal and capillary bleeding from the cut surfaces was noticed. The wound cavity was swabbed out with sterile normal saline solution and one gramme of powdered crystalline sulphanilamide was inserted before closure with Michel clips.

Results.—Figure I shows the results. From this it can be seen that all five controls died of gas gangrene in less than twenty-five hours, while of the treated groups only one died of this infection, and that seventy-six hours after inoculation. Three deaths from post-operative pneumonia

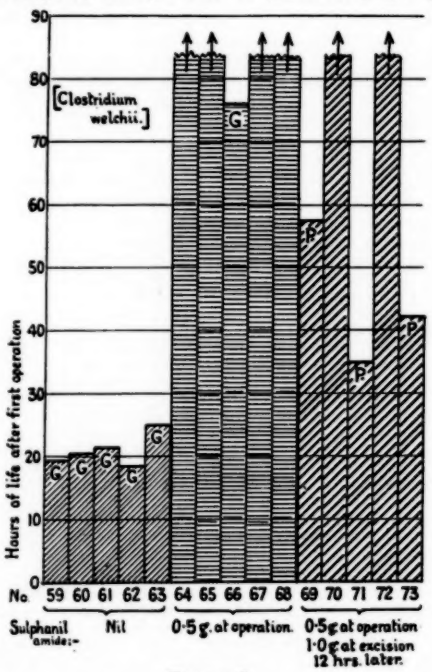

FIGURE I.
Results of the treatment of *Clostridium welchii* infections of guinea-pigs by sulphanilamide packed into the wound and by a combination of this treatment with surgical excision. G indicates death from gas gangrene, P indicates death from post-operative pneumonia, and an arrow indicates survival for more than ten days.

with extensive consolidation of the lungs occurred in the series in which excision was performed. There was no sign of gas gangrene in any of these animals, and in only one of the wounds could any organisms be found on a smear. In the six animals surviving after ten days, the only difference seen was that those in which the wound had been excised had a cleanly healed scar or a small superficial ulcer, whilst all the others in which the earth and dead muscle had not been removed, had subcutaneous abscesses under their wounds. Apart from these local lesions, all the surviving animals were quite healthy ten days after the original operation.

Statistical Analysis.—In the absence of treatment by a one-half gramme dose of sulphanilamide, the probability that the observed results are due to chance is 1/42. In the absence of treatment by two doses of sulphanilamide and excision of the wound the probability that the observed differences are due to chance is 1/252. These results are considered sufficiently definite to allow conclusions to be drawn from them.

Comment.—This work confirms that of Legroux,[15] who found that animals infected with *Clostridium welchii* could be saved by two applications of sulphanilamide to the infected wound, although oral therapy had little or no effect. A search of the literature has disclosed no other work on local therapy in this infection; but there are several reports of the results of oral administration. Henderson and Gorer[14] reported that, although sulphanilamide and sulphapyridine by mouth had a slight inhibitory effect on intradermal infection with washed *Clostridium welchii* bacilli, they had no effect on the infection caused by intramuscular injection of the organism. Singer[13] found that moderately good protection against infection with one minimum lethal dose of *Clostridium welchii* was given by all the sulphonamides; but when multiples of one minimum lethal dose were used the effects of oral therapy were very slight. Local application of sulphanilamide has been shown to be very effective in controlling infection with *Clostridium welchii*, and the results are much better than can be obtained by oral therapy alone. However, it is interesting to note that both Singer[16] and Stephenson and Ross[17] have shown that the slight beneficial effect of the oral administration of sulphonamide in this infection is greatly enhanced by serum therapy, apparently due to synergic action.

Clostridium septique.

The experiments with *Clostridium septique* were made as follows.

A cooked meat medium culture of *Clostridium septique* incubated for twenty-four hours at 37° C. was prepared from stock culture "L1" of the collection of the Institute of Medical and Veterinary Science, Adelaide. Culture "L1" corresponds to the National Collection of Type Cultures number 215, known as Weinberg 3.

In the first fifteen animals (numbers 40 to 58) the same operative procedure as detailed for *Clostridium welchii* was used; but after the muscle had been crushed the tissues were stripped from the superficial and deep surfaces of the ilium for a short distance and the bone in the neighbourhood of the iliac crest was fractured with a forceps. In the last five animals (numbers 108, 110, 111, 112, 113) the fracture of the bone was omitted so that excision of the wound twelve hours later could be more rapidly performed.

Results.—Figure II shows the results of this series of experiments. The control animals all died within twenty hours. Without excision the effect of the local implantation of 0·5 gramme of sulphanilamide was to double the length of life of the animals. When excision was carried out twelve hours after the original operation, one animal (number 113) died of gas gangrene, and then only after 113 hours. Three others were healthy and had well healed wounds after ten days. There was one death from post-operative pneumonia, but this animal survived for sixty-two hours.

Statistical Analysis.—In the absence of treatment by one-half gramme dose of sulphanilamide, the probability that the survival for more than thirty hours of most of the treated group is due to chance is 1/500·5. In the absence of treatment by two doses of sulphanilamide and surgical excision of the wound, the probability that the observed difference in mortality rate between this group and the controls is due to chance is 1/42. In the absence of excision and an additional dose of sulphanilamide, the probability that the difference in mortality rate between the two treated groups is due to chance is 1/273.

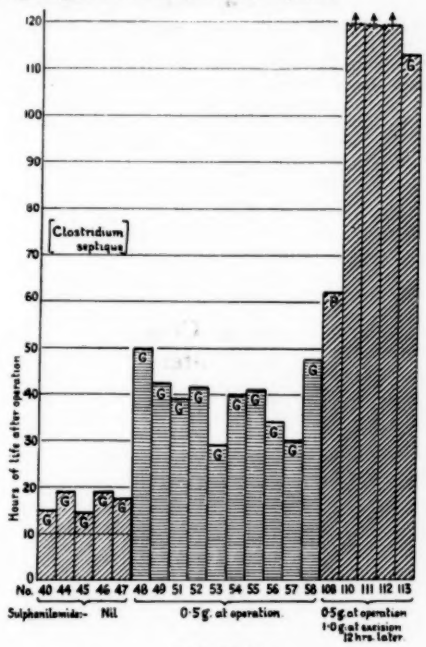
Hours of life after operation
Clostridium septique
No. 40 44 45 46 47 48 49 51 52 53 54 55 56 57 58 108 110 111 112 113
Sulphanilamide:- Nil
0·5 g. at operation
0·5 g. at operation
1·0 g. at excision 12 hrs. later

FIGURE II.

Results of the treatment of *Clostridium septique* infections of guinea-pigs by sulphanilamide packed into the wound and by a combination of this treatment with surgical excision. G indicates death from gas gangrene, P indicates death from post-operative pneumonia, and an arrow indicates survival for more than ten days.

Comment.—These results show that sulphanilamide applied locally exerted a definite delaying effect on the infection, and in combination with excision saved some animals. This result is much better than those recorded by Singer[16,17] and by Stephenson and Ross,[15] who used oral treatment with sulphanilamide and sulphapyridine. No other report of the local use of the sulphonamides in *Clostridium septique* infections can be found. Using oral therapy alone, Singer[13] found that no definite protection could be obtained in infections with this organism. This is in substantial agreement with the work of Stephenson and Ross.[17] Both these writers report that, as in *Clostridium welchii* infections, much better results were obtained by combined sulphonamide and serum therapy. All writers using sulphonamides by mouth in *Clostridium septique* infections agree that sulphapyridine is superior to sulphanilamide. It would be interesting to determine whether sulphapyridine would maintain its superiority when given locally.

Clostridium œdematiens.

The *Clostridium œdematiens* experiments were made as follows.

A 48-hour liver-piece broth culture of *Clostridium œdematiens*, strain Weinberg, Pasteur Institute, Paris, was supplied by Dr. A. W. Turner, Division of Animal Health and Nutrition, Council for Scientific and Industrial Research. Exactly the same experimental methods were employed as have already been described for *Clostridium welchii*.

Results.—As a glance at Figure III shows, the results were much less satisfactory. The sulphanilamide had a slight delaying effect on the rapidity of development of

the infection. Twelve hours after the inoculation excision was attempted; but a widespread gelatinous œdema of the subcutaneous tissue was found, and in spite of an extensive operation complete removal of diseased tissue was impossible. This severe operation, carried out on animals with a spreading gas gangrene infection, apparently hastened death. Pneumonia was probably the deciding cause of death of two animals (numbers 129 and 135). In both of these there was a gelatinous œdema of the subcutaneous tissue over the anterior abdominal wall, although the wounds looked comparatively healthy. These findings have been indicated by the letters "P.G." on the graph, and in the statistical analysis the animals are not considered to have survived the anaerobic infection.

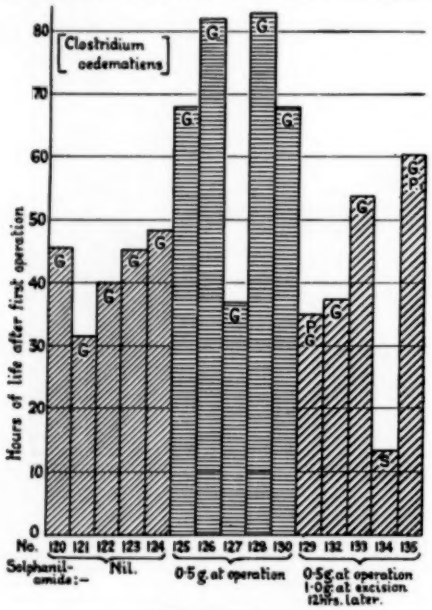

FIGURE III.

Results of the treatment of *Clostridium œdematiens* infections of guinea-pigs by sulphanilamide packed into the wound and by a combination of this treatment with surgical excision. G indicates death from gas gangrene, P indicates death from post-operative pneumonia, S indicates death from post-operative shock, and PG indicates that the animals died of pneumonia but had post-mortem signs showing some extension of the gas gangrene infection.

Statistical Analysis.—In the absence of treatment by one half-gramme dose of sulphanilamide, the probability that the survival of most of the treated animals for more than sixty-five hours is due to chance is 1/42. In the absence of treatment by excision and two doses of sulphanilamide the probability that the difference between the survival times of treated and control groups is due to chance is 1/4·5. In this group, therefore, the results are inconclusive.

Comment.—No other reports on the local treatment of *Clostridium œdematiens* infections with sulphonamides have been found. All other investigators (Stephenson and Ross[17] and Singer[15][16]) have found that sulphanilamide administered by the oral and parenteral routes had no retarding effect on infection with *Clostridium œdematiens*, and that it even sometimes accelerated the infection. Our results show a definite but slight delaying action on the progress of infection; but in general they confirm the fact that *Clostridium œdematiens* is much less susceptible to sulphanilamide than the other anaerobes.

Staphylococcus aureus.

The *Staphylococcus aureus* experiments were made as follows.

Organism.—A 24-hour broth culture of a strongly hæmolytic staphylococcus obtained from a carbuncle was used.

The routine wound was made and part of the ilium was crushed. No earth was placed in these wounds. Each wound was inoculated with two drops of the culture.

Results.—Forty-eight hours after operation there was a definite difference between the treated and untreated animals. All animals had some swelling in the region of the wound, but in the untreated group the mass was larger and fluctuant, and the surrounding skin was reddened. There were no deaths from this infection; but eight days after inoculation all animals, treated and untreated, had discharging abscesses in the region of the wound.

Comment.—The results from this small series are inconclusive, and merely suggest that the sulphanilamide had some delaying action in the growth of the organisms. It appeared that the infection lit up after the absorption of the half-gramme dose of sulphanilamide. The results with a mixed infection, which included the same staphylococcus, suggest that excision with repeated applications of the drug might have controlled the infection. Somewhat similar experiments were performed by Jensen, Johnsrud and Nelson[1] on a larger series of animals. Their results were rather indefinite, but suggested that sulphanilamide applied locally usually delayed the onset of infection and in some cases prevented it. Herrell and Brown[7] obtained striking results clinically by the local use of sulphathiazol in wounds infected with *Staphylococcus aureus*, and experimental work is required to confirm these findings.

Mixture of Organisms.

Experiments with a mixture of organisms were made as follows.

Unsterilized soil obtained from an old garden which had been cultivated with horse manure for many years was placed in each wound, along with a mixed culture containing the following organisms in roughly equal proportions:

(i) *Clostridium welchii.*—A 24-hour culture at 37° C. in cooked-meat medium from the heart blood of guinea-pig number 66, which had died seventy-six hours after infection with the original culture of *Clostridium welchii.*

(ii) *Clostridium septique.*—A 24-hour culture at 37° C. in cooked-meat medium from the heart blood of guinea-pig number 49, which had died forty-two hours after infection with the original culture of *Clostridium septique.*

(iii) *Staphylococcus aureus.*—A 24-hour broth culture at 37° C. of a hæmolytic strain from a carbuncle.

The operation was the same as that used in the *Clostridium welchii* series.

Results.—The striking efficacy of sulphanilamide in infection with this highly virulent mixture of organisms is evident in Figure IV. All control animals had died of gas gangrene in less than twenty-four hours. Four of the treated animals were alive and well at the end of eight days; one died of gas gangrene after 130 hours. The cause of death of the remaining five treated animals was pneumonia, with extensive pulmonary consolidation, and in none of these was there any evidence of extension of the gas-gangrene infection. It was intended to excise the wound in the control as well as in the treated animals; but examination of the former animals twelve hours after the infecting operation showed them to be too sick for excision to be attempted. In all of them the skin was crepitant over the whole left thigh and anterior belly wall.

Statistical Analysis.—In the absence of treatment by two doses of sulphanilamide and excision of the wound, the probability that the results are due to chance is 1/16,796. This highly significant figure, obtained by the use of a longer series than in the infections with single organisms, increases the reliability of the results of these last series. The same strains of *Clostridium welchii* and *Clostridium septique*, enhanced in virulence by animal passage, were used, and the deaths among the control animals indicated widespread distribution of both organisms.

Comment.—Infections with mixtures of known virulent organisms do not appear to have been investigated by other workers, though Singer[15][16] has tried the effect of sulphanilamide alone and in combination with serum on infections caused by the injection of a suspension of

garden soil. He found that, as in pure infections with *Clostridium welchii* and *Clostridium septique*, some protection was afforded by the combined use of sulphapyridine and a mixed antitoxic serum. Even with this combined treatment, however, his results are not nearly so good as those reported here. The results of this series of experiments reinforce the results of the investigations carried out with *Clostridium welchii* and *Clostridium septique*, in which the series were somewhat smaller.

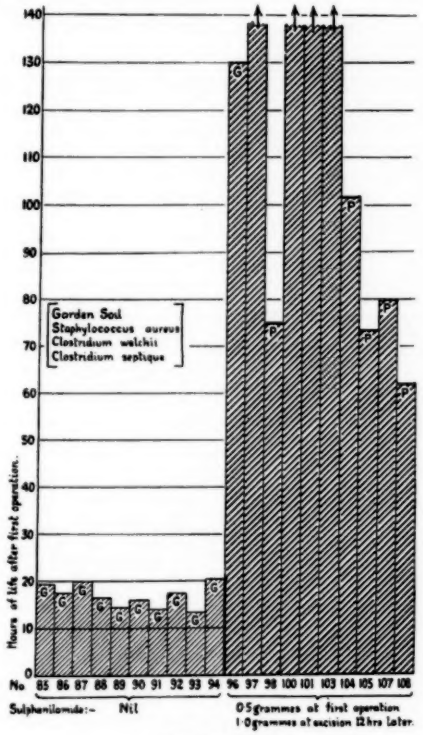
Garden Soil
Staphylococcus aureus
Clostridium welchii
Clostridium septique
Hours of life after first operation.
No 85 86 87 88 89 90 91 92 93 94 96 97 98 100 101 103 104 105 107 108
Sulphanilamide:- Nil
0·5 grammes at first operation
1·0 grammes at excision 12 hrs later.

FIGURE IV.

Results of the treatment of infections of guinea-pigs with a mixture of organisms—*Staphylococcus aureus*, *Clostridium welchii*, *Clostridium septique*, and garden soil—by packing of the wounds with sulphanilamide and surgical excision. G indicates death from gas gangrene, P indicates death from post-operative pneumonia, and an arrow indicates survival for more than ten days.

Streptococcus pyogenes.

The hæmolytic streptococcus is the most common cause of serious wound infection. That local therapy with sulphanilamide is very effective against this organism has already been shown by Nitti,[3] Levaditi,[2] Legroux,[5] and Herrell and Brown.[7] In view of all this evidence and of the well-known potency of orally administered sulphanilamide in streptococcal infections, we did not include a series of experiments with these organisms.

Summary of Results.

The experiments described have shown that locally administered sulphanilamide controls infections with *Clostridium welchii* and *Clostridium septique*, but has only a slight delaying effect on *Clostridium œdematiens*. The effect of local treatment in staphylococcal infections needs further investigation, but results suggest that progress of the infection is delayed. In combination with surgery it proved life-saving in infections with a virulent mixed culture.

There is a rather surprising contrast between these results and those obtained *in vitro* by Spray.[6] He found that there was an apparently specific selective inhibitory action on *Clostridium œdematiens* (*novyi*) and on *Clostridium septique*, while *Clostridium welchii* was scarcely affected.

Considering all investigations that have been carried out, we can say that sulphanilamide given locally has a powerful inhibitory effect on all the pathogenic bacteria commonly found in wounds, except *Clostridium œdematiens*, which is only slightly affected. There are indications that some of the other sulphonamides, especially sulphathiazol and sulphapyridine, would be still more efficient, particularly against *Staphylococcus aureus*.

Suggested Treatment for War Wounds.

Based on the results of these experiments and upon a study of all the available literature, the following suggestions for the treatment of war wounds are proffered.

Briefly, it is suggested that about 20 grammes of sulphanilamide be supplied to each soldier. This sulphanilamide should be poured into a wound immediately after the injury, either by the man himself or by a companion, and the first field dressing should then be applied in the usual way. On the casualty's arrival at the casualty clearing station, the wound should be excised and again packed with sulphanilamide, whether primary closure is attempted or not.

These suggestions need some elaboration. Work is necessary to determine the best form in which to supply sulphanilamide for local application. The powder is unsuitable, for it is somewhat sticky, does not pour readily, and is difficult to pack into a wound. It would probably be best to use small tablets or granules of the pure compressed powder.

When the local application of sulphanilamide is to be made by medical officers, various other methods may be used. Legroux[5] has worked at this problem and recommends the use of a powder insufflator for most wounds and of solid pencils of the compressed powder for insertion along bullet tracks. If for any reason attention to the wound is necessary, either at the regimental aid post or by officers of the field ambulance, then sulphanilamide should be again applied before the wound is redressed.

In order to cause a more rapid rise in the blood sulphanilamide concentration, oral administration of one of the sulphonamides at an early stage may be advisable. The issue of four tablets of sulphapyridine to each soldier would facilitate early administration.

Whether it will be possible after excision to close wounds which have received early local treatment with sulphanilamide will have to be determined by experience. Provided the wounds are filled with sulphanilamide before closure, this treatment may be found to be safe. This would represent a great advance, for primary closure and union by first intention must remain the surgical ideal.

If wounds are left open, repeated local application of the drug in powder form, as used by Levaditi, may be employed. Surgeons at the Mayo Clinic have used irrigation with a 0·5% solution and application of a paste for the purpose. The Winnett-Orr (Trueta) closed plaster treatment of open wounds has many practical advantages in wartime. It is suggested that if the treatment is used, it should be modified by packing of the wound with sulphanilamide before application of the plaster. This will delay the growth of organisms and may prevent that early burst of pyrexia which must represent a period of infection and invasion by pathogenic organisms with resulting tissue damage.

The main problem of wound management in wartime is to get the injured man to the surgeon before multiplication of organisms and invasion of tissue take place. Our experiments suggest that the immediate local use of sulphanilamide prophylactically in all wounds may go far towards the solution of this problem.

Need for Further Investigation.

Further experimental investigation on several points is required. The effects of local treatment by other sulphonamides and especially sulphapyridine and sulphathiazol on infection by all the common pathogenic bacteria must be studied.

When solid sulphanilamide is applied to a wound, the extent to which the paste or solution in the exudate extends into the further recesses of the wound must be determined, for it is in remote tissue pockets that infection is most likely to light up.

The necessity for combining oral administration with local treatment should be investigated, and dosage should be determined by blood sulphonamide estimations.

Clinical study should be carried out on all aspects of the subject, including convenient modes of application of the drug.

Summary.

An endeavour has been made to determine the value of the local application of sulphanilamide for the prevention and treatment of infection in wounds.

Previous experimental and clinical studies on local sulphonamide therapy have been briefly reviewed.

The properties of sulphanilamide and its effect on the growth of bacteria have been considered. In high concentrations the growth of most pathogenic bacteria is inhibited.

Experiments have been performed to determine the effect of sulphanilamide on animal tissues. Considered in conjunction with the work of other writers, they show that the drug is non-irritating to wound surfaces and body tissues generally, and that its application to open wounds is painless and causes no inflammation or interference with wound healing.

Implantation of sulphanilamide in a wound results in an almost saturated solution in the wound exudate for some days, and from this local depot slow steady absorption into the blood stream occurs.

Severe contused wounds have been inflicted on guinea-pigs, and different series of these wounds have been infected with *Clostridium welchii*, *Clostridium septique*, *Clostridium œdematiens*, *Staphylococcus aureus* and a mixture of organisms. The effect of locally administered sulphanilamide on these infections, alone and combined with surgical intervention, has been determined.

It has been found that sulphanilamide applied locally has a pronounced inhibitory effect on the growth in wounds of *Clostridium welchii* and *Clostridium septique*, a slight delaying action on *Clostridium œdematiens* and a probable delaying action on *Staphylococcus aureus*.

It was found possible by combining this treatment with surgical excision to save a high percentage in successive series of animals infected with *Clostridium welchii*, with *Clostridium septique*, and with a mixed culture containing these two anaerobes and *Staphylococcus aureus* with garden soil.

Based on this work and on the results of other investigators, a suggested treatment for war wounds has been developed, the routine and early local use of sulphanilamide being recommended as an important measure in preventing infection in wounds.

Lines for further investigation have been indicated.

Acknowledgements.

We desire to thank our Commanding Officer, Lieutenant-Colonel F. H. Beare, for permission to conduct these experiments. Our thanks are also due to Professor A. E. Platt, who has given us assistance in the provision of cultures and has maintained a critical interest throughout; and to Mr. E. A. Cornish for assistance with the statistical analysis of results.

We are indebted to Captain A. F. Hobbs, Officer Commanding the Camp Hospital, Woodside, for the provision of various facilities. The fact that we were able to carry out this work in camp is largely due to the inventiveness and industry of our laboratory assistant, Corporal J. F. Mayland.

The sulphanilamide used in these experiments was supplied by Drug Houses of Australia Limited.

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THE BIOCHEMICAL CHANGES IN PARALYTIC ILEUS AND INTESTINAL OBSTRUCTION.[1]

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(From the Walter and Eliza Hall Institute of Research in Pathology and Medicine.)

THE biochemical changes in paralytic ileus and intestinal obstruction have received a great deal of attention from previous workers. In 1914 Tileston and Comfort(1) found an increase in the non-protein nitrogen and urea nitrogen content of the blood in both conditions. A decrease in the blood chloride content was first noted by McCallum and his co-workers,(2) and in 1918 McCann(3) drew attention to the increase in the alkali reserve. These observations have since been confirmed in clinical and experimental studies by Louria,(4) Haden and Orr,(5) Gamble and McIvor,(6) and Hastings and his co-workers.(7)

In the present study a detailed clinical and biochemical investigation has been made of the changes which have occurred in four patients with paralytic ileus (Cases I, II, III and V) and in two with intestinal obstruction (Cases IV and VI). Determinations were made of the pulse rate, blood pressure, cell volume, blood urea, glucose and chloride contents, plasma protein content, alkali reserve and serum calcium content; the volume and chloride, calcium, urea and glucose content of the urine were also determined, and the chloride and calcium content of the vomitus. A record was also made of the

[1] This work has been carried out with the aid of a grant from the National Health and Medical Research Council.

volume of fluid lost by vomiting and of that taken by mouth and administered by intravenous infusion.

The existing methods of clinical biochemical analysis were modified so that all determinations could be carried out on six cubic centimetres of blood (Bick and Wood[8]).

Reports of Cases.

CASE I.—A.L., a male patient, aged twenty-two years, was admitted to hospital on April 20, 1939, and died on April 30, 1939. He had a history of melena nine years before, and two years before his admission to hospital he was treated for "ulcer" pain. An X-ray examination then revealed the typical deformity of a duodenal ulcer. The patient suffered from "ulcer" pain for six weeks, and three days before his admission to hospital he vomited blood; this was followed by severe melena.

On examination the patient was restless and pale. He was treated with morphine, transfusion of blood and intravenous infusion of saline solution; but bleeding continued, and an operation was performed on April 22, 1939, under light spinal anæsthesia. Morphine and hyoscine were given before operation. A small chronic ulcer was found on the posterior wall of the first part of the duodenum. Hæmorrhage had occurred from a vessel crossing the base of the ulcer. The ulcer was oversewn, the duodenum was divided above the ulcer and a gastro-enterostomy was performed. These procedures took two hours to complete. During the next twenty-four hours he was given a further transfusion of 600 cubic centimetres of blood, followed by 750 cubic centimetres of glucose in saline solution and 500 cubic centimetres of sodium lactate solution. His general condition appeared to be good.

On April 23, 1939, his condition was satisfactory, although he had vomited small amounts of brownish fluid during the night. The systolic blood pressure was 130 millimetres of mercury, the pulse rate was 90 per minute, and the hæmoglobin value was 63%; the alkali reserve was 44%, the plasma protein content 5·0%, the blood urea level 27 milligrammes *per centum*, the plasma sodium chloride content 550 milligrammes *per centum*, and the serum calcium content 8·5 milligrammes *per centum*. Glucose in saline solution (2,000 cubic centimetres) and sodium lactate solution (500 cubic centimetres) were given intravenously.

On April 24, 1939, the pulse rate, the respiration rate and the temperature had fallen; the systolic blood pressure was 130 millimetres of mercury, and the hæmoglobin value was 61%; the blood urea level was 39 milligrammes *per centum*, the plasma sodium chloride content 510 milligrammes *per centum*, the plasma protein content 4·6%, the alkali reserve 50%, the glucose content 110 milligrammes *per centum*, and the serum calcium content 8·6 milligrammes *per centum*. The Fouchet test failed to produce a reaction. He excreted 32·2 grammes of urea and 5·5 grammes of sodium chloride in 1,110 cubic centimetres of urine during twenty-four hours. The patient's condition appeared to be better, although he continued to vomit small quantities of material with the odour of gastric contents. An attempt to pass a stomach tube was frustrated by the distress of the patient. During the day 500 cubic centimetres of serum and 1,000 cubic centimetres of glucose in saline solution were given. The intravenous infusion was discontinued in the evening. The vomiting continued, and in twenty-four hours the patient vomited 1,910 cubic centimetres and had taken only 1,260 cubic centimetres of fluid by mouth.

On April 25, 1939, the hæmoglobin value was 63%, the blood urea level 32 milligrammes *per centum*, the plasma sodium chloride content 570 milligrammes *per centum*, the plasma protein content 4·7%, the alkali reserve 43%, and the glucose content 190 milligrammes *per centum*. The Fouchet test produced a reaction and the icteric index was 26. No obvious jaundice was present, although the urine was bile-stained. He excreted 30·1 grammes of urea and 4·5 grammes of sodium chloride in 1,050 cubic centimetres of urine in twenty-four hours. The patient was given a stomach washout and 570 cubic centimetres of fluid were removed. An enema produced a good result. The temperature was 100° F., the pulse rate was 90 per minute, and the respirations numbered 26 per minute. The tongue was moist, the abdomen was not distended and some flatus was passed. The intravenous infusion of fluid was recommenced during the afternoon and 2,500 cubic centimetres of glucose in saline solution and 1,000 cubic centimetres of sodium lactate solution were given intravenously in nineteen hours. The patient vomited 1,890 cubic centimetres of dark brown fluid in twenty-four hours.

On April 26, 1939, the pulse rate was 72 per minute, the systolic blood pressure 130 millimetres of mercury, and the hæmoglobin value 58%; the blood urea level was 28 milligrammes *per centum*, the plasma sodium chloride content 520 milligrammes *per centum*, the plasma protein content 6·0%, the alkali reserve 43%, the glucose content 90 milligrammes *per centum*, the serum calcium content 8·0 milligrammes *per centum*, and the icteric index 26. During the day the stomach was washed out four times and 1,200 cubic centimetres of fluid were removed. The returning fluid was slightly stained a brownish colour, and analysis showed that it contained a trace of blood, but no bile. An enema produced a good result; but melena was still present. The temperature was elevated in the evening and the pulse rate was 100 per minute. Vomiting continued during the night; 2,550 cubic centimetres of gastric contents were lost in twenty-four hours. During this time 800 cubic centimetres of glucose in saline solution and 1,000 cubic centimetres of glucose in Ringer's solution were given intravenously; 360 cubic centimetres of fluid were taken by mouth.

On April 27, 1939, the pulse rate was a little faster and the volume was diminished; the systolic blood pressure was 110 millimetres of mercury and the hæmoglobin value 60%. The stomach tube was passed every three hours and 1,770 cubic centimetres of fluid were aspirated in twenty-four hours. He was given 2,000 cubic centimetres of glucose in Ringer's solution and 1,000 cubic centimetres of 3% sodium chloride solution intravenously. Vomiting still continued, resulting in the loss of 2,310 cubic centimetres of fluid in twenty-four hours.

On April 28, 1939, the patient complained of a sudden severe pain extending all over the abdomen, radiating to the right shoulder, and in the right leg. On examination some collapse and tenderness were detected in the right iliac fossa. The hæmoglobin value was 62%, the systolic blood pressure 100 millimetres of mercury, the alkali reserve 53%, the plasma protein content 6·7%, the plasma sodium chloride content 530 milligrammes *per centum*, and the serum calcium content 8·9 milligrammes *per centum*; 390 cubic centimetres of urine containing 8·6 grammes of urea were excreted in the past twenty-four hours. The pain persisted and a further operation was performed under high spinal anæsthesia. At operation two perforations were found above a constriction in the anastomotic loop. There were approximately 1,800 cubic centimetres of bile-stained fluid in the peritoneal cavity. The perforations were closed and the constriction was straightened. During the operation 540 cubic centimetres of blood and 600 cubic centimetres of glucose in Ringer's solution were given intravenously.

On April 29, 1939, the pulse rate was 130 per minute, the systolic blood pressure 95 millimetres of mercury, the hæmoglobin value 68%, and the temperature 102° F.; the respirations numbered 30 per minute. No vomiting had occurred since the operation; jaundice was still present. Glucose in Ringer's solution was given intravenously; very little fluid was taken by mouth.

On April 30, 1939, the systolic blood pressure was 80 millimetres of mercury, the hæmoglobin value 45%, the alkali reserve 26%, and the blood urea level 145 milligrammes *per centum*. Death occurred shortly afterwards, following an acute attack of abdominal pain, which presumably was the result of a further perforation.

CASE II.—J.F., a male patient, was admitted to hospital on November 23, 1938, and discharged on December 23, 1938. An operation for perforated appendix with general peritonitis was performed on November 23, 1938. After the operation the patient was moderately collapsed, so an intravenous infusion of glucose in saline solution was commenced. The hæmoglobin value was 115%, the systolic blood pressure 100 millimetres of mercury, the blood urea level 47 milligrammes *per centum*, the alkali reserve 48%, the cell volume 50%, the plasma protein content 8·0%, the plasma sodium chloride content 510 milligrammes *per centum*, the glucose content 180 milligrammes *per centum*, and the serum calcium content 10·7 milligrammes *per centum*. He was given 3,960 cubic centimetres of glucose in Ringer's solution intravenously and 2,010 cubic centimetres of fluid were taken by mouth in twelve hours.

On November 24, 1938, the patient's condition was much better, so the infusion was discontinued. The hæmoglobin value was 108%, the cell volume 49%, the systolic blood pressure 120 millimetres of mercury, the alkali reserve 50%, and the plasma sodium chloride content 530 milligrammes *per centum*; the blood urea level had fallen to 23 milligrammes *per centum*, the plasma protein content to 6·2%, and the serum calcium content to 9·7 milligrammes *per centum*. During the preceding twelve hours 13·0 grammes of urea had been excreted in 1,380 cubic centimetres of urine. In spite of the intake of 35·6 grammes of sodium chloride the plasma chloride value had not risen, and only 0·8 gramme of sodium chloride was excreted in the urine; 2,460 cubic centimetres of fluid were taken by mouth in twenty-four hours.

On November 25, 1938, the patient was moderately well; the hæmoglobin value was 105%, the blood urea level 15

milligrammes *per centum*, the plasma protein content 6·2%, the plasma sodium chloride content 540 milligrammes *per centum*, the alkali reserve 47%, the glucose content 140 milligrammes *per centum*, and the serum calcium content 10·3 milligrammes *per centum*. A negligible amount of sodium chloride was still being excreted in the urine—0·9 gramme in 1,470 cubic centimetres.

On November 26, 1938, the abdomen had been distended for the past twelve hours, and during this time there had been persistent vomiting of 2,400 cubic centimetres of fæcal material. The alkali reserve had fallen to 32% and the plasma sodium chloride content to 430 milligrammes *per centum*, and the blood urea level had risen to 55 milligrammes *per centum*. Only small fluid stools had been passed. The passage of a stomach tube and the withdrawal of 5,220 cubic centimetres of gastric contents in twelve hours gave relief.

On November 27, 1938, the hæmoglobin value was 82%, the systolic blood pressure 140 millimetres of mercury, the cell volume 35%, and the alkali reserve 28%; the blood urea level had fallen to 33 milligrammes *per centum* and the plasma protein content to 5·4%; 23·1 grammes of urea were excreted in 1,440 cubic centimetres of urine in twenty-four hours. Gastric contents (7,470 cubic centimetres), fæcal in appearance, were aspirated in twenty-four hours. Fluids were taken well by mouth.

On November 28, 1938, it was recorded that for the past two days the abdomen had remained distended and the patient had gradually become weaker. He was given sedatives in the form of "Sodium amytal" and morphine. Glycerine enemata produced small fluid stools. During twenty-four hours aspiration yielded 6,750 cubic centimetres of clear gastric contents, and 7,400 cubic centimetres of glucose in Ringer's solution were given intravenously. The alkali reserve was 40%, the blood urea level 16 milligrammes *per centum*, the plasma protein content 5·1%, the plasma sodium chloride content 470 milligrammes *per centum*, and the serum calcium content 9·9 milligrammes *per centum*. The systolic blood pressure had risen to 170 millimetres of mercury. This was possibly due to the high intraabdominal pressure, which also caused some embarrassment of respiration.

On November 29, 1938, the abdomen was less distended; the patient's condition appeared to be generally improved and he was given a high protein fluid diet. The continuous intravenous therapy was stopped. The blood urea level was 23 milligrammes *per centum*, the plasma protein content 5·4%, the plasma sodium chloride content 460 milligrammes *per centum*, the serum calcium content 11·0 milligrammes *per centum*, and the hæmoglobin value 57%. The alkali reserve had risen to 56% and the systolic blood pressure had fallen to 140 millimetres of mercury. The stomach contents were aspirated every two or three hours.

On November 30, 1938, the stomach tube was removed, as no aspiration was required during the night, and after the frequent passage of large amounts of fæces the abdominal distension subsided. On December 1, 1938, the patient's condition continued to improve. The blood urea level was 20 milligrammes *per centum*, the plasma protein content 6·0%, the plasma sodium chloride content 450 milligrammes *per centum*, the alkali reserve 58%, and the serum calcium content 10·5 milligrammes *per centum*; 1·5 grammes of sodium chloride were excreted in 1,290 cubic centimetres of urine in twenty-four hours.

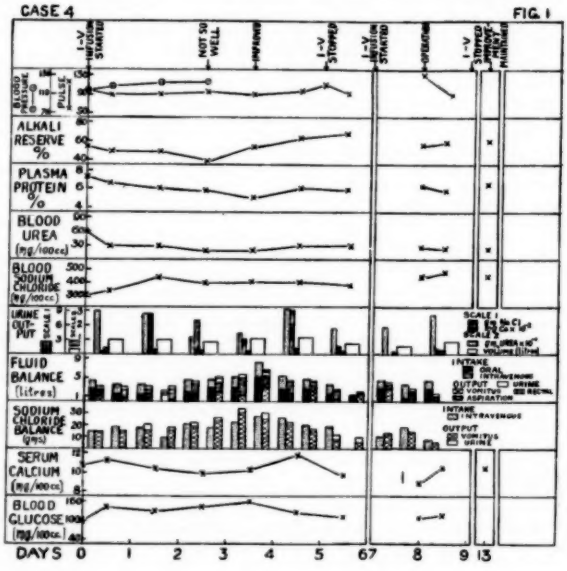
On December 5, 1938, the patient's condition was greatly improved; the cell volume was 40%, the plasma protein content 6·0%, the plasma sodium chloride content 470 milligrammes *per centum*, the alkali reserve 62%, the serum calcium content 11·1 milligrammes *per centum*, and the blood urea level 16 milligrammes *per centum*. The patient was eating well, but was unwilling to take a large amount of chloride.

On December 24, 1938, the hæmoglobin value was 93%, the systolic blood pressure 115 millimetres of mercury, the cell volume 45%. The plasma sodium chloride content had risen to 570 milligrammes *per centum* and the plasma protein content to 7·8%.

CASE III.—J.P., a female patient, aged forty years, was admitted to hospital on March 10, 1939, and discharged on April 12, 1939. Three days after an operation for a ruptured ectopic pregnancy and a curette the patient's abdomen became distended, and over a period of twelve hours during which 300 cubic centimetres of fluid were taken by mouth, she vomited 450 cubic centimetres of fluid. She was very pale and complained of abdominal pain; the abdomen was distended, but not tense. The systolic blood pressure was 110 millimetres of mercury, the hæmoglobin value 40%, the cell volume 24%, the blood urea level 45 milligrammes *per centum*, the alkali reserve 48%, the plasma protein content 7·5%, the plasma sodium chloride content 470 milligrammes *per centum*, the glucose content 100 milligrammes *per centum*, and the serum calcium content 9·7 milligrammes *per centum*. No chloride was excreted in the urine. Fluid was given intravenously, in the form of 600 cubic centimetres of 3% saline solution followed by 1,000 cubic centimetres of glucose in saline solution. Ten hours later the systolic blood pressure was 115 millimetres of mercury, the hæmoglobin value 37%, the cell volume 22%, and the alkali reserve 52%. The blood urea level had fallen to 29 milligrammes *per centum* and the plasma protein content to 6·6%. The plasma sodium chloride content had risen to 570 milligrammes *per centum*. The bowels had been well opened and the patient's abdomen was less distended. A transfusion of 780 cubic centimetres of blood was begun. At the beginning of the transfusion the patient was restless, with some air hunger. After the transfusion the systolic blood pressure had risen to 130 millimetres of mercury, the hæmoglobin value to 55%, the cell volume to 28%, and the plasma sodium chloride content to 620 milligrammes *per centum*. The blood urea level was 30 milligrammes *per centum*, the alkali reserve 56%, the glucose content 140 milligrammes *per centum*, the plasma protein content 6·9%, and the serum calcium content 9·0 milligrammes *per centum*. The transfusion was followed by the intravenous infusion of 1,000 cubic centimetres of glucose in saline solution.

On March 15, 1939, the patient's colour was much better and she was brighter. The alkali reserve had risen to 65% and the serum calcium content to 10·6 milligrammes *per centum*. The plasma sodium chloride content was 620 milligrammes *per centum*, the plasma protein content 6·8%, the cell volume 26%, the blood urea level 24 milligrammes *per centum*, and the glucose content 120 milligrammes *per centum*. The concentration of sodium chloride in the urine was 0·02%. On March 16, 1939, the patient's condition was still improving; the average concentration of sodium chloride in the urine was still small—0·07%. By April 12, 1939, the patient was taking a normal diet. The plasma protein content had risen to 7·6%, the plasma sodium chloride content was 560 milligrammes *per centum*, and the concentration of sodium chloride in the urine was 0·24%. The blood urea level was 23 milligrammes *per centum*, the alkali reserve 56%, and the serum calcium content 9·9 milligrammes *per centum*.

CASE IV.—E.F., a male patient, was admitted to hospital on June 7, 1938, and discharged on July 25, 1938 (see Figure I). On June 9, 1938, after the patient had suffered abdominal pain for two days, a gangrenous appendix was removed. Drainage was considered unnecessary and the wound was closed. The following day he vomited on several occasions and the abdomen became distended.



On June 11, 1938, vomiting continued, with some slight abdominal pain, and examination revealed a distended abdomen with visible coils. The tongue was dry; the systolic blood pressure was 120 millimetres of mercury, the pulse rate was 100 per minute, the temperature was 100° F.,

and the respirations numbered 20 per minute. The cell volume was 53%, the alkali reserve 54%, the plasma protein content 7·4%, the blood urea level 59 milligrammes *per centum*, the blood sodium chloride content 310 milligrammes per centum, the glucose content 100 milligrammes *per centum*, and the serum calcium content 10·8 milligrammes *per centum*. The intravenous infusion of glucose in Ringer's solution was commenced and a stomach tube was passed.

On June 12, 1938, the systolic blood pressure was 125 millimetres of mercury, the pulse rate 90 per minute, and the temperature 99° F.; the blood urea level had fallen to 31 milligrammes *per centum*, the alkali reserve to 48%, and the plasma protein content to 6·7%. The cell volume was 50%, the blood sodium chloride content 350 milligrammes *per centum*, the glucose content 140 milligrammes *per centum*, and the serum calcium content 11·3 milligrammes *per centum*. He excreted 480 cubic centimetres of urine in twelve hours; the urea concentration was 4·4%, and 0·3 gramme of sodium chloride was present in the urine. The patient vomited 1,050 cubic centimetres of fluid, and 1,620 cubic centimetres of gastric contents were aspirated in twelve hours. Fluids (2,250 cubic centimetres) were given by mouth, and 1,650 cubic centimetres were given intravenously.

On June 13, 1938, the patient was still continuing to vomit material that was now fæculent. The systolic blood pressure was 135 millimetres of mercury, the pulse rate 90 per minute, the alkali reserve 48%, the blood urea level 27 milligrammes *per centum*, and the glucose content 130 milligrammes *per centum*; the plasma protein content had fallen to 6·2% and the serum calcium content to 10·6 milligrammes *per centum*; the blood chloride content had risen to 440 milligrammes *per centum*; 6·2 grammes of sodium chloride and 22·3 grammes of urea were excreted in 1,290 cubic centimetres of urine in twenty-four hours. Glucose in Ringer's solution (3,960 cubic centimetres) was given intravenously, and 3,480 cubic centimetres of water and orange drinks were taken by mouth. Aspiration yielded 3,870 cubic centimetres of gastric contents, and 1,590 cubic centimetres of fluid had been vomited.

On June 14, 1938, the patient was not so well; the alkali reserve had fallen to 38%, the plasma protein content to 5·8%, and the blood chloride content to 392 milligrammes *per centum*, although the average sodium chloride concentration in the urine was 0·74%. By aspiration 5,760 cubic centimetres of fluid were obtained and 780 cubic centimetres of gastric contents were vomited in twenty-four hours; 39·1 grammes of sodium chloride were lost in this way. The intravenous infusion was still being continued; 3,460 cubic centimetres had been given in twenty-four hours and 3,630 cubic centimetres of fluid were taken by mouth. In the evening the patient was drowsy and vomiting everything taken. The respirations were deeper than normal, although the rate was still 20 per minute. A milk and treacle enema preceded by the injection of 0·5 gramme of acetylcholine produced little result, but some flatus was passed. The rate of flow of the Ringer's solution was increased and 3,000 cubic centimetres were given in the next twelve hours.

On June 15, 1938, the patient's condition appeared to be better and a milk and treacle enema produced an excellent result. Despite this, the abdomen was more distended. The pulse rate was 90 per minute and the pulse was of good volume. Some pus containing *Bacillus coli communis* was now being discharged from the wound. The cell volume was 44%, the blood urea level was 19 milligrammes *per centum*, the glucose content was 160 milligrammes *per centum*, the blood chloride content was 410 milligrammes *per centum*, and the serum calcium content was 10·3 milligrammes *per centum*. The alkali reserve had risen to 54%; 11·0 grammes of urea were excreted in 1,020 cubic centimetres of urine in twenty-four hours. A glycerine enema with sennatin produced no result. During this period of twenty-four hours 7,920 cubic centimetres of fluid were taken by mouth, 6,380 cubic centimetres were aspirated and 3,240 cubic centimetres were lost by vomiting. The aspirated fluid had now changed from dark brown to golden brown in colour.

On June 16, 1938, the pulse rate was 96 per minute, the cell volume 48%, the blood chloride content 414 milligrammes *per centum*, and the glucose content 120 milligrammes *per centum*. The alkali reserve had risen to 63%, the plasma protein content to 6·0%, the serum calcium content to 11·9 milligrammes *per centum*, and the blood urea level to 30 milligrammes *per centum*. The amount of urea in the urine was increased, 28·3 grammes being excreted in twenty-four hours. The abdomen was much less tense; a milk and treacle enema, preceded by acetylcholine, produced a good result. On this day 7,920 cubic centimetres of fluid were taken by mouth; but 5,790 cubic centimetres of gastric contents were lost by vomiting and aspiration. Milk and egg white were given in an endeavour to raise the serum protein content. During the night the patient was mentally confused, the temperature rose to 103° F. and the pulse rate was 120 per minute. The intravenous infusion was discontinued, the stomach was then washed out and the tube was removed. The patient did not vomit again for five hours, but then vomited 750 cubic centimetres of light brown fluid. The abdomen was then comparatively normal and borborygmi could be heard. The pulse rate was 84 per minute, the temperature 97° F., the alkali reserve 68%, the plasma protein content 5·9%, the blood urea level 29 milligrammes *per centum*, the glucose content 120 milligrammes *per centum*, and the blood sodium chloride content 390 milligrammes *per centum*; the serum calcium content had fallen to 9·6 milligrammes *per centum*. After being given morphine the patient appeared to be better, but in the afternoon he became anxious and restless.

On June 18, 1938, the pulse rate was 108 per minute; 960 cubic centimetres of fluid were vomited in two hours, the stomach tube was reinserted, and the intravenous infusion of glucose in saline solution was recommenced. There was a good bowel action attended by severe colicky pain, and a ladder pattern with strong peristaltic waves could be seen on the abdominal wall. During this day 4,110 cubic centimetres of fluid were taken by mouth and 3,000 cubic centimetres of glucose in saline solution were given intravenously; but 5,400 cubic centimetres of gastric contents were lost by vomiting and 2,400 cubic centimetres by aspiration.

On June 19, 1938, laparotomy was performed and the small bowel was found to be dilated above a small pelvic adhesion in the appendiceal region. Below this the bowel was collapsed. After operation the pulse rate was 130 per minute, the alkali reserve 56%, the plasma protein content 6·2%, the blood urea level 27 milligrammes *per centum*, the blood chloride content 460 milligrammes *per centum*, and the serum calcium content 8·9 milligrammes *per centum*. Only 120 cubic centimetres of fluid were vomited, but 5,910 cubic centimetres of fluid were aspirated in twenty-four hours; 2,910 cubic centimetres of glucose in Ringer's solution were given intravenously.

On June 20, 1938, the pulse rate was 90 per minute. The infusion was now discontinued. The bowels were opened during the day. On June 21, 1938, the patient's condition was improved; there was no vomiting and the stomach tube was removed. The systolic blood pressure was 120 millimetres of mercury, the pulse rate 88 per minute.

On June 22, 1938, the improvement was maintained, the pulse rate was 86 per minute, the cell volume 40%, the alkali reserve 50%, the plasma protein content 5·5%, the blood urea level 36 milligrammes *per centum*, the blood sodium chloride content 420 milligrammes *per centum*, and the serum calcium content 9·6 milligrammes *per centum*. The excretion of chloride in the urine was still low; 0·7 gramme of sodium chloride was excreted in twenty-four hours. The bowels were well open. The patient was given fluids and light food with a high protein content.

On June 24, 1938, the pulse rate was 88 per minute; the plasma protein content had risen to 6·4%, the alkali reserve to 60%, and the blood chloride content to 460 milligrammes *per centum*. The improvement was steadily continued, and when the patient was discharged on July 25, 1938, the plasma protein content was 7·0% and the blood sodium chloride content 490 milligrammes *per centum*.

CASE V.—M.F., a female patient, was admitted to hospital on February 6, 1939, and died on February 13, 1939. Twenty-four hours after an operation had been performed (on February 8, 1939) to relieve an obstruction caused by adhesions, the result of previous gynæcological operations, the patient's abdomen was distended and she vomited frequently. The vomitus consisted of bile-stained fluid. The bowels had not been opened. In the evening the patient's abdomen was more distended and she complained of thirst. Saline solution given by the rectum was retained. The systolic blood pressure was 112 millimetres of mercury, the temperature was 100° F., and the pulse very rapid; the alkali reserve was 54%, the blood urea level 50 milligrammes *per centum*, the plasma protein content 7·7%, the plasma sodium chloride content 520 milligrammes *per centum*, the serum calcium content 10·3 milligrammes *per centum*, and the cell volume 53%; the concentration of chloride in the urine was 0·02%. An intravenous infusion of 1,000 cubic centimetres of 3% sodium chloride solution was commenced. This was followed by 1,000 cubic centimetres of glucose in saline solution. A stomach tube was passed.

On February 10, 1939, the patient was drowsy, with a raised respiration rate; the abdomen was not rigid, but some distension was still present. The intravenous administration of glucose in saline solution, which had been discontinued for the previous six hours, was resumed. The cell volume was 50%, the serum calcium content 9·8 milli-

grammes *per centum*, the plasma protein content 7·5%, and the glucose content 190 milligrammes *per centum*. The alkali reserve had fallen to 34% and the blood urea level to 28 milligrammes *per centum*; the plasma sodium chloride content had risen to 600 milligrammes *per centum*. The urinary chloride concentration was 0·04% and urea concentration 2·1%. After 1,500 cubic centimetres of glucose in saline solution and 1,000 cubic centimetres of sodium lactate solution had been given, the intravenous infusion of fluid was discontinued, as there was some œdema at the site of injection and in the bases of the lungs. During the day 1,080 cubic centimetres of gastric contents were aspirated.

On February 11, 1939, the cell volume was 40%, the blood urea level 34 milligrammes *per centum*, the plasma sodium chloride content 610 milligrammes *per centum*, and the glucose content 180 milligrammes *per centum*; the alkali reserve had risen to 40%, and the serum calcium content to 11·3 milligrammes *per centum*; the plasma protein content had fallen to 6·4%. The bowels were opened and the abdomen appeared to be less distended. During this period of twenty-four hours 3,600 cubic centimetres of gastric contents were aspirated and 750 cubic centimetres of urine were excreted.

On February 12, 1939, the cell volume was 43%, the alkali reserve 42%, the plasma protein content 6·7%, the plasma sodium chloride content 600 milligrammes *per centum*, the glucose content 160 milligrammes *per centum*, and the serum calcium content 10·7 milligrammes *per centum*; the blood urea level had risen to 75 milligrammes *per centum*. The urea concentration in the urine was 1·1% and the concentration of chloride 0·04%; 780 cubic centimetres of urine were excreted in twenty-four hours, and 2,850 cubic centimetres of gastric contents were aspirated during this time. The intravenous infusion of glucose in saline solution was resumed; but improvement did not occur and death resulted on the following day.

CASE VI.—E.L., a female patient, was admitted to hospital on August 24, 1939, and died on August 27, 1939. Eleven days after a hysterectomy had been performed, the patient's abdomen became distended; this distension was relieved by an enema. Twenty-four hours later the patient was very distressed after taking food and vomited clear fluid. Another good result was obtained with an enema.

On August 25, 1939, enemata failed to produce any relief, and the patient was restless and complained of nausea and thirst; the tongue was slightly moist and the abdomen was distended, but not rigid. The systolic blood pressure was 110 millimetres of mercury, the pulse rate was 112 per minute, the alkali reserve 62%, the plasma protein content 6·4%, the plasma sodium chloride content 420 milligrammes *per centum*, the blood urea level 26 milligrammes *per centum*, the cell volume 35%, the glucose content 80 milligrammes *per centum*, and the serum calcium content 9·1 milligrammes *per centum*. There was no chloride in the urine. The intravenous infusion of glucose in Ringer's solution was commenced. The patient was very nervous and a stomach tube could not be passed; 660 cubic centimetres of fluid were vomited during the afternoon. During seven hours 1,000 cubic centimetres of glucose in Ringer's solution and 2,000 cubic centimetres of 3% saline solution were given. The systolic blood pressure was then 112 millimetres of mercury, the pulse 90 per minute, the plasma sodium chloride content 540 milligrammes *per centum*, the blood urea level 28 milligrammes *per centum*, the plasma protein content 5·8%, and the serum calcium content 10·6 milligrammes *per centum*; the alkali reserve had fallen to 45%. Under a spinal anæsthetic aided by the administration of a small amount of ether laparotomy was performed and the obstruction, caused by bands and adhesions, was relieved. The intravenous infusion of glucose in Ringer's solution was continued.

On August 26, 1939, the pulse rate was 180 per minute, the blood urea level 34 milligrammes *per centum*, the glucose content 160 milligrammes *per centum*, and the cell volume 34%; the alkali reserve had fallen to 36%, the plasma chloride content to 430 milligrammes *per centum*, and the plasma protein content to 5·4%. During twelve hours 420 cubic centimetres of urine were excreted; the urea concentration was 0·75% and chloride was still absent from the urine; 420 cubic centimetres of fluid had been taken by mouth, and a further 3,000 cubic centimetres of fluid had been given intravenously. During the next seven hours 2,000 cubic centimetres of sodium lactate solution were given; but there was no improvement, and the patient died on August 27, 1939.

Discussion.

In these conditions, as a result of the extensive loss of fluid, food and salts by vomiting and by aspiration, there is a deficiency of sodium chloride associated with a pronounced diminution of chloride in the urine, some degree of acidosis and a depletion of the plasma protein content. The blood urea level is generally slightly elevated. As food and fluids cannot be given by mouth to these patients owing to the persistent vomiting, intensive intravenous therapy must be used as the chief method of correcting these changes, of supplying nourishment and of compensating for the excessive loss of fluids. Intravenous infusions of 5% or 10% glucose in normal saline solution, 5% glucose in Ringer's solution, 3% saline solution, tenth molar sodium lactate solution and plasma or serum have to be given, according to the nature of the biochemical changes.

Sodium Chloride Content.

In these cases there was a pronounced decrease in the chloride content of the blood accompanied by a diminution of chloride in the urine. In five cases the plasma sodium chloride content ranged from 420 to 550 milligrammes per 100 cubic centimetres of plasma, when the observations were commenced. In the remaining case (Case IV) the blood sodium chloride content was 310 milligrammes per 100 cubic centimetres. The fall in the blood chloride content follows the excessive loss of chloride caused by the severe and prolonged vomiting, and it has been shown experimentally by White and Bridge[9] that the fall in the chloride content of the blood and tissues corresponds to the amount of chloride lost in the urine and vomitus. This loss may be replaced by the intravenous infusion of normal or hypertonic 3% saline solution. Haden and Orr,[10] however, found depletion of chloride in a case of intestinal obstruction when there was no vomiting. As a result of experimental work on animals, they consider that some chloride is utilized in the course of the increased tissue destruction which takes place.

In the present series one patient (Case IV) lost by vomiting 59·6 grammes of sodium chloride in twenty-four hours. In this case 5% glucose in Ringer's solution was administered intravenously, and although it was possible to maintain a positive sodium chloride balance there was no elevation of the blood chloride content. It was more satisfactory in such cases to infuse small volumes of 3% saline solution to correct the deficiency (Cases V and VI). The infusion of hypertonic saline solution, however, frequently causes no increase in the chloride content of the blood, although amounts of sodium chloride adequate to replace all that is lost are given, and the excess chloride is not excreted in the urine. In one case (Case II) 45·3 grammes of sodium chloride were lost in forty-eight hours by vomiting and by aspiration of the gastric contents. During this time 104·3 grammes of sodium chloride were given by the intravenous infusion of two litres of 3% saline solution and 3·9 litres of Ringer's solution containing glucose; 2·5 grammes of sodium chloride were excreted in the urine and the plasma chloride level remained unchanged. Falconer and Lyall[11] have noted a pronounced variation in the response to saline administration in cases of obstruction, and suggest that the tissue reserves are increased before the blood chloride level is raised.

Alkali Reserve.

The alkali reserve was found to be diminished in five cases. The value obtained varied from 44% to 54% when the investigations were commenced. The majority of previous workers, however, have noted an increase in the alkali reserve in cases of obstruction. The administration of large quantities of fluid, either intravenously or by mouth, was found to relieve the acidosis in Cases III and IV. In other cases (Cases I, II and V) the alkali reserve continued to fall with the persistent vomiting, in spite of liberal administration of fluid, and in one case (Case II) it rapidly fell from 47% to 28% as the result of the vomiting of more than two litres of gastric contents. Tenth molar sodium lactate solution was also given to three patients (Cases I, V and VI) to relieve the acidosis, and in one case (Case I) the alkali reserve was raised from 43% to 53% in forty-eight hours by the infusion of one litre of this solution.

Plasma Protein Content.

The continuous intravenous infusion of large volumes of physiological fluids caused a decrease in the percentage of protein in the plasma. The diminution in protein was generally accompanied by a decrease in the cell volume percentage (Cases II and IV). This indicates that some of the decrease in protein percentage was attributable to an increase in the blood volume. Usually the protein depletion was small, the plasma protein concentration being greater than 6% (Cases III, IV and V). This depletion was corrected by a liquid diet rich in protein, which could be taken with comfort by the patient in a few days (Cases II and IV). In Case I, in which the obstruction had been preceded by repeated gastro-intestinal hæmorrhage before operation, the plasma protein content was 5·0% at the commencement of the investigation; in twenty-four hours it fell to 4·6%, when an intravenous infusion of 500 cubic centimetres of serum was given. This increased the plasma protein content to 6·0% in two days.

The determination of the plasma protein content should be made at regular intervals during prolonged cases of ileus and obstruction, as serious depletion of protein associated with the replacement of chloride and the infusion of large volumes of fluid may lead to œdema of the tissues.

Blood Urea Level.

The elevation of the blood urea level in cases of intestinal obstruction has been observed by other workers. In the present series the blood urea level was found to be slightly raised in Cases II, III, IV and V. The concentration of urea varied from 40 to 60 milligrammes per 100 cubic centimetres of blood. Kaufman[12] considers this rise to be due to the diminished circulatory volume and the increased destruction of the tissues. Falconer and Lyall[11] have noted that an increased blood urea content is associated with a high excretion of urea in the urine, indicating an increase in the endogenous protein metabolism.

It was found that the blood urea level fell rapidly soon after the infusion of fluid was commenced (Cases II, III, IV and V). The fall in the blood urea level was accompanied by excretion of urea in the urine. In one case (Case III) the blood urea level fell from 59 to 31 milligrammes *per centum* in twelve hours. During this period 21·0 grammes of urea were excreted in 480 cubic centimetres of urine of an average urea concentration of 4·4%. No impairment in the ability of the kidney to concentrate urea was observed in the present series.

Serum Calcium Content.

The serum calcium content remained within normal limits in practically all cases. It appears to drop as a result of the excessive loss of fluid from the alimentary canal. In some cases, in which the serum calcium content was low or falling, Ringer's solution was given intravenously with benefit (Cases I, II and IV).

Glucose Content.

In the present series the glucose content of the blood was always less than 200 milligrammes *per centum*. The concentration of glucose in the urine was always so small that it could not be detected by Fehling's test. It appears that the body is able to utilize the large amounts of glucose (200 to 300 grammes were given per day in Cases II and IV) which are given intravenously as the main source of nourishment (Figure I).

Fluid Intake.

To replace the fluid lost from the alimentary canal and to maintain an adequate output of urine large volumes of fluid must be given intravenously, and also by the mouth as orange drinks, barley water *et cetera*. In Case IV over 32 litres of physiological fluids were given in nine days. There were no signs of œdema. A record of the intake and output of fluid enables a sufficient volume to be infused to maintain a positive fluid balance. Falconer and Lyall[11] consider that the undoubted benefit of the administration of saline solution in cases of obstruction is due to the correction of dehydration rather than to the increase of the blood chloride content. However, it has been shown experimentally by White and Bridge[9] and other workers that the administration of water or isotonic glucose solution to correct dehydration is not sufficient, and that both sodium chloride and water are necessary to prevent a fatal alteration in the chemistry of the blood and tissues.

Summary.

1. A detailed investigation of the biochemical changes which occur during paralytic ileus and intestinal obstruction was made in six cases.

2. There was found to be a deficiency of sodium chloride associated with a diminution of chloride in the urine, a depletion of plasma protein content, and some degree of acidosis. The concentration of urea in the blood was somewhat above normal.

3. The effects of the intravenous administration of large volumes of physiological fluids are discussed.

4. The determination of the biochemical changes indicated the choice of fluids for intravenous infusion—for example, hypertonic saline solution, tenth molar sodium lactate solution, plasma or serum.

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Reviews.

MODERN DIETARY TREATMENT.

THE current emphasis upon the constructive possibilities of medical science is in itself recognition of the fact that universal practical application of the principles of nutrition is necessary in order to achieve full health in any community, and to treat the diseased members with success.

During the past ten or fifteen years the whole concept of nutrition has been revolutionized. The Calorie determination of dietetic adequacy has been superseded by the idea of

[1] "Modern Dietary Treatment", by M. Abrahams, M.A., M.Sc., and E. M. Widdowson, B.Sc., Ph.D.; Second Edition; 1940. London: Baillière, Tindall and Cox. Crown 8vo, pp. 413. Price: 10s. 6d. net.

balanced nutritional sufficiency—an advance made possible only by the rapid progress made in our investigation of vitamins and mineral metabolism and by the great improvements that have been brought about in methods of food analysis. Simultaneously with this advance has come the realization that one of our greatest problems now concerns the spread and the practical application of this new knowledge. Abrahams and Widdowson have made a valuable contribution to the solution of this problem in "Modern Dietary Treatment"[1] the second edition of which has just been published.

The book should prove especially useful, firstly, because of the excellent tables of food values, which are arranged to facilitate the planning of accurate and varied diets both in health and disease, and secondly, for the number of recipes which have been included with the particular aim of suiting the several different types of diet.

In the revised edition recent advances in the dietary treatment of disease have been considered and the text has been brought up to date. Thus diabetic diets for use with protamine zinc insulin have been added, and the dietary management of gastric ulcer and hyperchlorhydria has been revised. A diet for jejunostomy is also included with instructions for its administration. The section dealing with the treatment of Addison's disease by low potassium diets has been amplified, and a low phosphorus diet for hypoparathyreoidism is described. Ketogenic diets, on the other hand, have been omitted, since they are now almost obsolete.

The book as a whole is in keeping with the modern tendency to regard the primary dietary requirements of the body in disease as fundamentally the same as those of a healthy body; that is, to secure adequate nourishment by supplying sufficient of all essential food factors.

This book should prove extremely helpful to doctors, nurses, and all others interested in dietetics.

HEARING AND EQUILIBRIUM.

IN the preface of his book "Hearing and Equilibrium" H. Macnaughton-Jones states that its object is to give a brief account of the essential mechanism of both hearing and the maintenance of equilibrium in a manner which will be easily understood even by those who have the special knowledge neither of the ear nor of physics.[1]

In a great measure the author has achieved his object; but we consider that some knowledge of physics is necessary to understand his theories, and the author himself recognizes this, for he states in a later paragraph in the preface that there are short passages in refutation of the two misconceptions, which are obviously intended for those versed in the subject and may prove tedious to others.

We agree that the study of the ear, which ought to be one of the most fascinating studies, has been avoided in a great measure under the general impression that it is a difficult and abstruse subject. Presentations of the physiology of the ear have been uninteresting on the whole and explanations difficult to follow.

In this mongraph an effort is made to present the subject so that the reasoning can be understood by those not possessing exceptional knowledge. The author admits that his account of the mechanism of the ear is almost revolutionary and functions are attributed to various parts at total variance with those commonly assigned to them. He maintains that his statements are made strictly in accordance with recognized physical laws or are supported by irrefutable experimental evidence which can easily be verified. The functions of the different structures are interdependent, he states.

The author's theory that "in the ear, wave motion is directed to the membrane over the *fenestra rotunda* and that the basilar membrane and associated structures convert the cochlea into what may be regarded as a series of resonators, or preferably as a graduated resonator, capable of responding to an infinite gradation of frequency" is not new. However, the author has presented his subject matter in a new light and for this he deserves our thanks. While it is not possible to demonstrate that the mechanism is utilized, it is possible, the author claims, to demonstrate that such a mechanism is present, and that it possesses a property hitherto unsuspected, namely, that it would possess the necessary variations in frequency, without submitting its structures to tension.

A theory is put forward concerning the course that sound waves take in the ear. It has been long recognized that the tympanic membrane with the chain of ossicles acts merely as an accommodating apparatus, and physicists and physiologists have for a long time propounded theories in keeping with this present one—to mention but one the Zund-Burguet. However, the explanation of the author fits in more with the anatomical characteristics of the shape of the inner portion of the external auditory meatus, the drum head, the floor of the middle ear and the position of the *fenestra rotunda*. The diagrams presented show that it is the *fenestra rotunda* which is subject to the concentrated efforts of the column of air projecting inwards from the inner end of the external canal, whereas the *fenestra ovalis* is away from its path in what would correspond to a deep sound shadow. In addition, it is protected from the minor disturbances which ordinarily travel into a sound shadow from the periphery of the object producing the shadow.

While the main part of the book is devoted to the question of hearing, the subject of equilibrium has not been entirely neglected. One of our main objections to Dr. Macnaughton-Jones's work is that he appears to have placed too much reliance upon models and conclusions which he has drawn from these models, for it is rather claiming too much to argue that because certain results are obtained from models they must be those associated with a living organ. By offering this criticism we do not disparage the author's work, for we consider that the book should be studied by all interested in the subject, from the medical student to the established aurist. We recommend the book.

SURGERY OF THE HAND.

MR. HANDFIELD-JONES, in "Surgery of the Hand", has attempted to meet a very real need.[1] The subject looms so large in casualty practice and the success of treatment means so much to the patient that a concise and practical treatise is to be welcomed. We feel, however, that the scope of the book could have been confined to injuries and infections without impairment of its usefulness. The small section devoted to "other surgical conditions" is notable only for a brief but adequate account of glomus tumours. One or two diagrams of surface markings would have been of practical value.

The major part of the book is devoted to infections, which are presented clearly and dogmatically, mainly along traditional lines. Due emphasis is placed upon prophylaxis as a social and surgical problem. The advocacy of active movement from the moment drainage is instituted is a departure from the advice of Kanavel. Ring anæsthesia for the incision of paronychia is recommended without reference to its risks. Henry's ulnar approach to the mid-palmar space and the ulnar bursa, and the relation of the ulnar artery to secondary hæmorrhage in the forearm are illustrated. The adverse effect of secondary infection on the prognosis in tenosynovitis is wisely emphasized. A very useful section is devoted to the mobilization of stiff fingers, including corrective splinting and major operative procedures.

When the seventh edition of Kanavel's book appeared in 1939 an appreciation and evaluation of sulphanilamide in hand infections was not yet possible; and Mr. Handfield-Jones has been able to take advantage of the knowledge gained since then.

The chapter on fractures is largely drawn from Mr. Watson-Jones's monograph. Operation is recommended for late cases of mallet finger. For the difficult fractures of the neck of the fifth metacarpal reduction and fixation with the joint at a right angle appear to be the solution. Operation for irreducible dislocation of the thumb should be a capsulotomy under vision. A subungual hæmatoma can be extremely painful, and the relief which follows evacuation through the nail should perhaps have received mention.

The account of foreign bodies would have been enhanced by even more emphasis on the need for asepsis and by the inclusion of a method of precise localization, as for example, by the use of radiologically opaque guides.

On the subject of tendon injuries some pertinent generalizations are made. Immediate suture of an ensheathed tendon is both futile and dangerous, whereas a free tendon is sutured at once unless associated with a fracture. We should have enjoyed reading more of the authors' views regarding technique and after-treatment, and also something of tendon grafting. The principles of Bunnell's incisions would have been worth inclusion.

The author is to be congratulated for including injuries and infections of the hand in one volume. New books from England have an additional significance these days and we look forward with confidence to the next edition.

[1] "Hearing and Equilibrium", by H. Macnaughton-Jones, M.B., B.Ch., B.A.O., R.U.I.; 1939. London: Baillière, Tindall and Cox. Foolscap 4to, pp. 136, with 48 illustrations. Price: 7s. 6d. net.

[1] "Surgery of the Hand", by R. M. Handfield-Jones, M.C., M.S., F.R.C.S.; 1940. Edinburgh: E. and S. Livingstone. Imperial 8vo, pp. 149, with 95 illustrations, some of which are in colour. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, FEBRUARY 1, 1941.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

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THE THOMAS SPLINT IN THE FIRST-AID TREATMENT OF FRACTURE OF THE FEMUR.

In the second volume of "The Australian Army Medical Services in the War of 1914-1918", Colonel A. Graham Butler declares that with the exception of treatment of wounds by excision and primary suture, no greater advance in wound treatment was made in the war than the use of the Thomas splint in the front line to prevent shock. He publishes three pictures of a demonstration at the old advanced dressing station at Ypres of three stages in the application of the Thomas splint. On previous occasions attention has been drawn in these pages to the inordinate length of time that was allowed to elapse before methods proved to be reliable and recommended by the highest authorities were adopted as routine procedures by the army medical authorities. Among these must be numbered the first-aid use of the Thomas splint for fracture of the femur. Graham Butler tells us that it was in December, 1914, that the late Robert Jones, in a special report to the War Office and in a special article in *The British Medical Journal*, commended the Thomas splint for the transporting of wounded men with fractured femurs. Incidentally it may be recalled that Thomas offered his splint to the French army in the Franco-Prussian war and that the offer was not accepted. And as long ago as 1887 R. F. Tobin, in discussing gunshot fractures of the lower limbs in war, said that no splint on which the limb did not rest by its own weight gave satisfactory support when for any purpose the patient had to be lifted; the Thomas splint, he pointed out, fufilled these conditions.[1] At a discussion on the treatment of wounds involving fracture of the femur, held in February, 1915, under the auspices of the Australian Voluntary Hospital stationed in the Boulogne area, Sir Alexander MacCormick, consulting surgeon of the area, "emphasized the influence of the method of transport from the front on the outcome of the case. He suggested that the field ambulances at the front should be supplied with the Thomas splint, 'as it can be applied immediately and would obviate a lot of subsequent movement' ". Writing in this journal on October 22, 1932, George Bell states that Sir Alexander MacCormick actually bought and took across to France early in 1915 the first Thomas knee splints used for transport purposes in the war. By the end of 1915 the Thomas splint was in use in many units in France, but Butler writes that it was not until the battle of Messines, in 1917, that the splint "came fully into its kingdom".

It was a far cry from 1917 to 1870, and it is not to the credit of army surgeons that all this time had to pass before the Thomas splint came finally into "its kingdom". Still this has been achieved, and we may be quite certain that in the present world war the lesson learned so painfully in the first-aid treatment of fractures of the femur will not be forgotten. But this is not the object of the present discussion; the question that we wish to raise is whether the knowledge of the value of the Thomas splint bought of bitter experience in the last war is being applied to the needs of civil practice. The subject has been discussed on more than one occasion at medical gatherings in the past; unless some effective action can be taken it bids fair to become one of those perpetually recurrent topics that begin and end with words, the words sometimes taking the form of a pious resolution. Fracture of the shaft of the femur in civil life is generally the result of a serious accident; whether the fracture is simple or compound, it is likely to be accompanied by shock, and this shock may be increased to an alarming extent by injudicious or unnecessary movements. Apart from the pain that may be caused by the movement of a fractured limb, sight must not be lost of the anatomical arrangement of the blood vessels and of the ease with which they may be injured by the fragments of the fractured bone. These facts are well known to all medical practitioners, but if they are known to ambulance officers and others who have to undertake the first-aid treatment of injured persons, they are not always fully appreciated by them. It is this lack of appreciation that is really responsible for any inadequate treatment that is adopted. Readers are referred to THE MEDICAL JOURNAL OF AUSTRALIA of October 22, 1932, for a discussion that followed the reading of a paper by George Bell, and to the issue of November 24, 1934, for another, not less important though perhaps more spirited, that arose from a paper by J. C. Storey. At a recent meeting of the New South Wales Branch of the British Medical Association, reported in the present issue, R. Angel Money made statements that have impelled us to take up the question once more. As will be seen from the report, he said that ambulance officers did not seem to realize the importance of the Thomas splint. He had seen patients arrive at the Royal North Shore Hospital of Sydney in a Thomas splint; the ambulance officers took the patient to the X-ray room in a splint and then took off the splint because it was theirs and they had to take it away. The patient was "dumped" here and had to be lifted two or three times after that without the splint. In view of these statements and of those made by J. C. Storey, H. R. G. Poate and others at previous discussions, it appeared that a useful purpose might be served if information was collected from different parts of Australia in regard to the extent to which the Thomas splint was used in the first-aid treatment of fracture of the femur. With this object a circular letter

[1] Frederick Watson, "The Life of Robert Jones", 1934, page 153.

was written to the medical superintendents of hospitals in Brisbane, Sydney, Melbourne, Adelaide, Perth, Hobart and Launceston. Reference was made in this letter to the statements made at the recent Sydney meeting and the superintendents were asked whether they would state what proportion of patients brought to their institutions with fracture of the femur had had a Thomas splint applied at the time of the injury, and if this splint was applied by ambulance officers, what arrangements were made in regard to the disposal of the splint in which the patient's limb was slung. A reply was received to every letter. The General Superintendent of the Brisbane and South Coast Hospitals Board writes that each ambulance in Brisbane is equipped with a modified Thomas splint fitted with an apparatus for the application of extension. When a fractured femur is suspected this splint is applied by the ambulance officers. When the patient is admitted to the Brisbane Hospital the splint remains with the patient and the ambulance receives in its place a splint from a number stocked by the hospital. At Sydney the position appears to be curious. The General Superintendent of the Royal Prince Alfred Hospital writes that ambulances carry a Thomas splint and triangular bandages. Every patient with a fracture of the femur or a suspected fracture of the femur has a Thomas splint applied at the time of the injury. Spare splints and triangular bandages are kept in the casualty department, and these are substituted for those used by the ambulance officers and are taken away in the ambulance. The Medical Superintendent of Sydney Hospital states that patients with obvious fractures of the femur are usually brought by the ambulance with the limb slung in a Thomas splint; the splint belongs to the ambulance and is usually taken away by the officers. The General Medical Superintendent of the Prince Henry Hospital and the Medical Superintendent of Saint Vincent's Hospital, Sydney, report that patients with fracture of the femur are not brought to the hospital with a Thomas splint applied. All the medical superintendents of hospitals in the other States reported that the Thomas splint was not used by ambulance officers; several stated that a Thomas splint was usually found applied to the limb of a patient admitted from another institution or sent in by a private practitioner.

If ideal conditions prevailed throughout Australia, all hospitals would be able to make reports similar to those from Brisbane and from the Royal Prince Alfred Hospital, Sydney. There is no reason why these ideal conditions should not prevail. The failure in many centres cannot be laid entirely at the door of the ambulance organizations. Ambulance officers must look for guidance in first-aid procedure to the medical profession. After the war of 1914-1918, officers of the Australian Army Medical Corps should have found it simple to carry into civilian life the obviously sound procedures that they had learned in the field: there is no large regular army in this country and practically every officer on demobilization returned to civil practice. The trouble is that in the army routine procedures are adopted as a result of specific orders; in private civilian practice there is no one to say that this or that shall be done and each man is more or less a law to himself. The first-aid use of the Thomas splint in fracture of the femur will not be adopted throughout Australia unless it becomes someone's business to see that the desirability for its adoption is presented to ambulance organizations in the right way, and unless medical men are available and willing to act as instructors in the use of the splint. There are only two bodies in Australia that can readily approach surgeons in every State on such a matter; one is the Federal Council of the British Medical Association in Australia and the other is the Royal Australasian College of Surgeons. In urging that early consideration should be given to this matter, we would direct attention to the thirteenth appendix in Butler's "official history". This is a reproduction of instructions issued to the second Australian Casualty Clearing Station in October, 1918, for the front line application of the Thomas splint and gives "drill by numbers" that could easily be taught to ambulance officers throughout the Commonwealth.

Current Comment.

THREADWORMS.

DESPITE the frequency of threadworm infestation in children, and indeed in adults, there seems to be little clear appreciation among practitioners of the nature of this problem and the reliability of the various accepted means of controlling it. In a letter in this journal of June 1, 1940, A. Jefferis Turner reminded us that the infestation is an important question for all who treat children. There has been no reliable survey of the extent of the infestation among Australian children, but recent reports in Canada and America show that approximately 30% of school children are affected. The symptoms caused by threadworms are but vaguely understood by many. All sorts of complaints are attributed to this cause without adequate proof, and, as Turner points out, treatment for worms is all too often ordered by doctors and pharmacists purely as a speculation. Recent surveys by M. J. Miller and colleagues[1] and by F. J. Brady and W. H. Wright[2] agree that anal pruritus, nervousness and disturbed sleep are frequent symptoms. Miller found that nocturnal enuresis was much more common among infected children than among a control group, but teeth-grinding was not. Brady and Wright found enuresis just as common among their controls. The general health of the infected children was not good, and treatment produced a marked improvement in weight and appetite, and caused dark circles to disappear from under the eyes. In girls vaginitis was frequently found and it was a common experience to see the worms actually migrating into the vagina. These writers have no doubt at all that a threadworm vaginitis exists and indeed is common. In their survey they could find no evidence, however, that abdominal pain or appendicitis, acute or chronic, had any relationship to worm infestation. Anal irritation varied in intensity from a hardly perceptible sensation of the worms migrating through the anus to a most intense and intolerable pruritus, which the authors attempt to explain as an allergic skin response to some product elaborated by the worms. They felt certain that it was too severe to be caused simply by the mechanical irritation of the worms.

One difficulty in the control of this infestation is that of knowing when cure is complete, for the common methods of examining stools for the ova of parasites is of little use. Infestation takes place through the mouth. About a fortnight later the worms have attained maturity, and the gravid females migrate through the anus and lay their eggs on the perianal skin. The eggs are not laid in the bowel and so the number present in the stools is very limited. A more effective method is the taking of swabs from the perianal region. The method commonly in

[1] *The Canadian Medical Association Journal*, November, 1940.
[2] *The American Journal of the Medical Sciences*, September, 1939.

use by American and Canadian workers is the "NIH" swab. The swab consists of a glass rod tipped with a small square of "Cellophane". This rod is encased in a glass tube for convenience and for protection from infection in handling. In use the "Cellophane" tip is stroked over the perianal region, and the threadworm eggs if present will usually adhere to it. The "Cellophane" is then removed, mounted between a glass slide and cover slip and examined for ova. This method is used to establish the original diagnosis. The recommended criterion of cure is seven successive "negative" swabs at intervals of a few days, commencing two weeks after the course of treatment is completed.

The reason for such an exacting criterion becomes apparent when the possibilities of immediate reinfestation are appreciated. If all the worms are not cleared from the bowel, the cycle of maturation and reinfestation will soon be completed and the condition become as bad as ever. Reardon[1] has counted the number of ova found in gravid female worms. The mean of the counts in twenty worms was 11,105 ova. Another important factor often responsible for reinfestation is the frequency with which several members of a family are infested. Wright and Cram[2] have pointed out that it is useless to treat individuals in a household in which other individuals are left untreated, though infested. The abundant opportunities for reinfestation are indicated by the immense number of ova found above, and by the experience of Nolan and Reardon[3] who were able to recover threadworm ova from objects at all levels in all rooms in infested households, thus indicating the possibilities of air-borne infection. It is not only towels, washers, underclothes, soap *et cetera* that harbour the danger, but almost any object at all. Under moist warm conditions these ova remain viable for about two weeks.

It is plain then that the treatment of threadworms is not the simple matter that many people think. It is not just a matter of giving a few worm powders and watching the motions until no more worms are seen. The ideal threadworm anthelmintic should be safe enough to give over a sufficient period to allow desiccation of ova in the environment, either continuously or in courses repeated at short intervals, and it must be highly effective, cheap and easily administered without much trouble to the family. Most of the affected families are in the lower economic ranges, and have neither time nor money for tedious and expensive treatment. Wright and Cram survey the value of commonly used measures. Of the anthelmintics given in a single dose tetrachlorethylene is regarded as the most effective. Santonin given in repeated doses is regarded as being of little value. Enemas of hexylresorcinol, quassia, soapsuds or saline solution are thought to have a limited value, but are unpleasant and must often be continued for many weeks. Anal ointments are only an adjuvant. Anal plugs, designed to prevent the migration of the female and so the completion of the life-cycle, are probably of no value at all. Some of these treatments are frankly dangerous, some unnecessarily tedious, and all are shown by the "NIH" swab to fall far short of the necessary degree of effectiveness. The need for a cheap, safe and effective threadworm anthelmintic is very real. Those who claim that nothing but personal cleanliness is necessary must have their faith shaken by the knowledge of how widespread are the ova in infected households.

Both Wright and Brady[4] and Miller and his colleagues have recently made strong claims for gentian violet. It is safe if given in the recommended doses and cheap, and is shown by the "NIH" swab to be effective in 90% of cases. That means it is very much nearer to the ideal anthelmintic than anything that has been used previously. Miller gave the drug in doses of three-tenths of a grain three times a day to children between the ages of six and nine years, and half a grain to children of ten to thirteen years. Brady and Wright gave half a grain three times a day to adults, and to persons under the age of sixteen years, one-sixth of a grain per year of apparent age per day in a single dose. Some were given the drug in a single course lasting for ten days. Others had two courses each of eight days and separated by an interval of seven days. There was little difference in the effect. The drug was given in tablets covered by a water soluble coating of such a thickness that it dissolved in about four hours, and therefore liberated the drug in the small bowel. Mild toxic symptoms like nausea and vomiting occurred in about one-third of the patients, but were seldom severe enough to necessitate discontinuance of the treatment. Diarrhœa, mild abdominal pain, headache, dizziness or lassitude occurred in a few instances. Strict follow-up examinations on these patients by means of the "NIH" swab indicated that gentian violet is quite the most effective drug treatment yet available for threadworms. It should be accompanied, of course, by the usual measures of personal and household hygiene.

These reports are so emphatic and the drug is so easily and cheaply obtained that it is to be hoped that it will be extensively tried by Australian practitioners. But let it be tried, as any new therapy should be tried, with an honest and critical mind, willing to recognize and admit success or failure.

[1] *Public Health Reports*, June 17, 1938.
[2] *American Journal of Diseases of Children*, December, 1937.
[3] *Journal of Parasitology*, April, 1939.
[4] *The Journal of the American Medical Association*, March 9, 1940.

CARCINOMA OF THE BREAST IN A GIRL TEN YEARS OF AGE.

IN the November 7, 1940, issue of *The New England Journal of Medicine*, J. B. Sears and M. J. Schlesinger record the occurrence of an adenocarcinoma, grade II, in the breast of a white girl aged ten years. The lump in the breast had been present for six years. At the time of examination it was hard, irregular, sharply circumscribed and not tender; it was adherent to the overlying nipple, but was movable posteriorly. It measured 3·0 by 2·5 by 2·5 centimetres, and was removed by surgical operation. The pathological report is reproduced in full, and typical microscopic sections are reproduced. Sears and Schlesinger refer to two other reports of the occurrence of breast cancer before puberty, one in a girl of eleven and the other in a girl of twelve years. Apart from the clinical value of this case, it is of interest because of the work of Loeb and others on the relationship between carcinoma and œstrogenic hormones.

MATERNITY ALLOWANCES.

THE Acting Commissioner of Maternity Allowances has issued from Canberra his annual statement showing the number of claims for maternity allowances granted and rejected and the cost of administration for the twelve months ended June 30, 1940. In the terms of the *Maternity Allowance Act*, 1937, the payment of allowances is subject to an income limit of £247 in respect of births occurring on or after January 1, 1938. An additional £13 is added to this sum in respect of each previous living child under fourteen years of age, the maximum being £338. The amount of the allowance is £4 10s. when there is no previous living child under fourteen years of age, £5 when there are one or two previous living children under fourteen, and £7 10s. when there are three or more such children.

The total number of maternity allowances paid during the year under review was 77,352; in 68,870 cases the mother was attended by a doctor, and in 8,482 she was not. During the year 6,779 claims were rejected, 5,447 of them because of an excess of income and 1,002 because claims were withdrawn or not completed. The total amount paid in claims for the whole Commonwealth was £416,964. The cost of administration was £17,181. Since maternity allowances were first paid in October, 1912, 3,193,894 claims have been paid and 67,607 rejected.

Abstracts from Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Pertussis Antitoxin in Rabbits.

D. G. EVANS (*The Journal of Pathology and Bacteriology*, July, 1940) continued his work on pertussis by investigating the production of pertussis antitoxin in rabbits and the neutralization of pertussis, parapertussis and bronchiseptica toxin. Dried toxin, prepared according to the author's standards, was used in such dilution that 0·2 cubic centimetre gave no skin reaction on normal rabbits, and they were then given weekly injections until they had received 7·0 cubic centimetres. The production of antitoxin was first ascertained by means of intradermal doses of toxin of descending dilution. It was found that while a normal rabbit showed a reaction to toxin diluted to 1 : 640, the immunized rabbits did not react to dilutions of 1 : 2·5; this showed a high degree of immunity. The rabbits were then bled and the serum was tested for its protecting power against lethal doses and against skin necrotizing effects. The three specimens of serum tested varied a little, but all showed considerable antitoxic activity. They also showed agglutinating properties, and this was separate from the other effect. Further tests against parapertussis and bronchiseptica organisms gave similar results; this fact suggested that they should be grouped together. Subcutaneous injection of toxin was more successful in inducing immunity in these rabbits than intravenous injection. The author discussed his results and their variance from those observed on human subjects.

A Simple Culture Medium for General Use.

G. P. GLADSTONE AND PAUL FILDES (*The British Journal of Experimental Pathology*, August, 1940) describe a simple culture medium for general use, without meat extract or peptone. The necessary nitrogen is supplied by two fractions of casein, one obtained by acid hydrolysis, the other by tryptic digestion. Glutamine and a watery extract of yeast constitute the other two sources of essential growth substances. The medium is buffered by sodium glycerophosphate. Full details of the method of preparation of the basic medium are given, the optimum quantities are stated, and methods of storage are described. The solid basic medium is designated by the term "CCY agar" (casein casein yeast agar), and the preparation of various additional substances which can be added to enrich it for special purposes is also described. Without the agar a satisfactory substitute for nutrient broth is also obtained. One or other of the modifications of "CCY agar" has been found to support growth of all organisms of medical interest able to grow on meat media. Glucose is added to give maximum growth of hæmolytic streptococci and pneumococci. For *Corynebacterium diphtheriæ* the authors added peptic sheep blood and cystine, and this addition gave good differentiation between the different colonial forms and satisfactory appearances with Neisser stain. The liquid medium without glycerophosphate and with one-tenth the original amount of yeast was able to replace peptone water as the basic medium for sugars in the fermentation reactions. The authors also describe a method for growing pneumococci on "CCY agar" with glucose, in an atmosphere of 5% carbon dioxide. Culture plates are placed in tins with press-down lids, in which there is a tube of eight millilitres of 25% hydrochloric acid. Into this a marble chip about 0·7 gramme is dropped just as the lid is placed on the tin and the evolved carbon dioxide is trapped. The authors believe that this medium will be of use firstly on account of its low cost; further, the few modifications necessary to fit it for routine use in a laboratory will simplify work for a diminished staff, the basal solutions have been proved to keep almost indefinitely, and the ingredients are not likely to be unobtainable under war conditions.

Suprarenal Gland and Diphtheritic Toxæmia.

E. HOLMES AND H. LEHMANN (*The Journal of Experimental Pathology*, August, 1940) write on the relationship of lesions of the suprarenal gland to diphtheritic toxæmia. The work of many investigators shows that there is a *prima facie* case for the supposition that in diphtheria the brunt of the damage falls on the suprarenal gland. Experiments have been carried out on glucose tolerance, on the removal of lactic acid from the blood stream, and on the metabolism of liver slices from animals into which varying doses of diphtheria antitoxin had been injected. Considerable variations from normal have been found. The authors thought that removal of both suprarenal glands from normal animals should produce similar results if the theory that the suprarenal glands are damaged by the diphtheria toxin is correct. They devised a two-stage operation, in which the right suprarenal gland was first removed from rabbits, after which they recovered well; a week or more later the left gland was removed; the animals were then in poor condition, and if they were needed for longer than twenty-four hours injections of an active cortical extract were necessary. The results of the tests showed unchanged glucose tolerance, and the liver slices formed carbohydrate in a normal manner from added lactate. The authors think that the explanation of disorders of carbohydrate metabolism found in association with diphtheritic toxæmia is not to be found in its effect on the suprarenal glands, but more probably in damage to the liver cells.

Virus as a Cause of Pneumonia in the Mongoose.

JOHN M. WEIR AND FRANK L. HORSFALL (*The Journal of Experimental Medicine*, November, 1940) describe the recovery of a virus causing pneumonia in the mongoose, from patients suffering from an unusual form of pneumonitis. The patients were examined in December, 1938; throat washings were taken in nutrient broth and blood serum was obtained at the onset of the illness as well as one month later. First attempts to infect a large number of varieties of small laboratory animals from the washings uniformly failed, and then the work was transferred to Jamaica, where the wild mongoose could be trapped in large numbers. Intranasal inoculation of the animals in pairs was carried out, material from four different patients being used, and in each case after the inoculation of two cubic centimetres of washings the animals developed pulmonary lesions of uniform type. The lesions presented varying degrees of plum-coloured gelatinous consolidation, from which the virus could be recovered and further animals could be infected. Control animals inoculated with sterile saline solution did not develop any lesions, so that the possibility of the activation of a latent pneumotropic virus was controlled. A contact experiment between inoculated and normal mongooses showed that the infection could be transmitted from animal to animal, four out of five uninoculated contacts showing typical lesions at autopsy. Serial passage of a lung filtrate was achieved, Berkefeld filters being used. The viability of the virus was also tested, and it was able to resist glycerination, freezing and long standing at room temperature. The virus was also propagated on the chorio-allantoic membrane of the chick, both from throat washings and from infected mongoose lung. Neutralization tests were made with mixtures of virus and patient's serum, and while the serum obtained in the early days of the human disease did not give protection, that obtained from convalescent patients and also that from convalescent mongooses did protect laboratory animals. Cross neutralization tests with patient and mongoose serum and known strains of influenza virus failed to suggest any identity between this virus and that of ordinary influenza. The authors conclude that they isolated from patients suffering from a clinical syndrome termed acute pneumonitis a virus which was capable of producing pulmonary consolidation in the mongoose, that the virus was the cause of the pneumonitis, and that it was different from other viruses known to cause infections of the respiratory tract in man.

Survival of Pneumococci with Sulphapyridine.

ERWEN NETER (*The Journal of Infectious Diseases*, September-October, 1940) has investigated the survival of pneumococci in empyemic fluids, and the effect of sulphapyridine upon their viability. The author had previously made clinical observations on the failure of the drug in some cases of empyema, and had contrasted this failure with its success in preventing the development of experimental rabbit empyema. Specimens of exudate fluid were obtained from six patients with pneumococcal empyema who had been treated with the drug, and estimations were made of the amount of the drug present in the exudate; portions of the fluid were then incubated *in vitro*, and subcultures were made to find the length of time during which the organisms survived. It was found that they lived up to twenty days in fluid containing as much as 4·8 milligrammes *per centum* of sulphapyridine, and similar results could be obtained from fluid in which there was no drug, while in some specimens of fluid containing much less, the survival time was under eight days. Specimens of fluid from untreated patients were also mixed with known concentrations of the drug, up to 50 milligrammes *per centum*, and were subcultured for as long as five days. The organisms were still alive after eighteen hours in the strongest solution, but when the experiments were repeated with fluid from the same patient a week later, the organisms were much more resistant than earlier in the disease. The results were not

consistent, even in such a small series, and the author thinks that while the effectiveness of sulphapyridine in other diseases cannot be denied, its value in pneumococcal empyema is by no means proved.

Medium for Isolation of Bovine Tubercle Bacilli.

ALFRED G. KARLSON (*The Journal of Infectious Diseases*, July-August, 1940), during a study of bovine tuberculosis, paid special attention to a suitable medium, easily prepared for a large-scale cultivation of the bovine tubercle bacillus. Since glycerine was very commonly used in the cultivation of the human type, and since it had been suggested that the glycerine might inhibit the growth of the bovine type, four simple media were prepared both with and without added glycerine (4%). Fresh material was obtained from 36 slaughtered cattle which had reacted to the tuberculin test during life, so that "wild" cultures, or primary isolations could be contrasted with laboratory strains acclimatized by many subcultures to growth on artificial media. The results obtained were striking. A simple egg yolk agar prepared with beef extract showed growth on 96·8% of tubes without glycerine after three to four weeks' incubation, whilst only 12·1% of those with glycerine showed growth after four to six weeks' incubation. The total number of tubes in the experiment was 124. Similar but less striking figures were obtained for the other media without glycerine, in contrast to the same with glycerine. The author holds that in the cultivation of pathogenic material in which bovine tubercle bacilli may be present, a medium which does not contain glycerine should always be included.

HYGIENE.

Nutrition and Health in Pasadena.

H. BORSOOK AND W. L. HALVERSON (*American Journal of Public Health*, August, 1940) report that the diets of fifty families chosen at random as to income were intensively studied at Pasadena, and a year later clinical tests were applied to the 151 adults and 72 children (under 14) concerned. Very complete laboratory vitamin estimates were made. The lower limit of cost for an adequate diet was sharply defined at $2.08, but well-informed groups got down to $1.50 per week per person for a first class diet. About half the families had less than the 3,000-Calorie intake recommended, yet underweight occurred equally whether the Calories were above or below the standard. All received over 45 grammes of protein, half or more of which was animal in origin and seemed adequate. Practically all subjects received 25% or more of the Calories as fat and the majority one-third. Calcium in the majority was inadequate, that is, under 660 milligrammes daily. Iron was adequately taken. Only eight families received adequate amounts of vitamin B_1 (over 4,000 international units daily). One-third received less than the recommended figure, 500 international units. Probably 750 international units are desirable; only, one-quarter received this. Physiological stresses, pregnancy, fever, stress of work, make greater demands. Peanut butter and whole-wheat bread and cereal containing middlings and germ keep up the vitamin B_1 supplies. The vitamin B_1 finding was adopted as covering the vitamin *B* complex requirement. Thanks to leafy vegetables, vitamin *C* seemed adequate. In spite of a fairly good supply of fruit and vegetables, vitamin *A* was as a rule short, while one-third were not receiving adequate vitamin B_1. Vitamin *D* was not included. The clinical tests showed 6% infected tonsils and two cases of mild rickets. Height and weight variations were not unusual or limited to any form of diet. Hæmoglobin was satisfactory. Out of 56, 17 were free from caries; no correlation was found between freedom from caries and a high calcium intake or a high expenditure on food. The authors consider that the effect of a suboptimal diet can be determined only by tests carried out over a number of years.

The Effects of Cigarette Smoking.

W. A. HIESTAND, H. J. RAMSEY AND D. M. HALE (*The Journal of Laboratory and Clinical Medicine*, July, 1940) describe the effects of cigarette smoking on the metabolic rate, heart rate, oxygen pulse and respiratory rate of a group of subjects comprising heavy smokers, light smokers and abstainers. Immediate effects and also the effects over a longer period of time were noted. The metabolic rates were determined by means of a McKesson metabolar. A group of 38 subjects was tested; smoking caused an increase in metabolic rate in 82% of the subjects, a decrease in 13%, while 5% showed no change. In some cases the maximum effect on smoking one cigarette was reached immediately, but in others it was delayed as long as forty-five minutes. The first rise in metabolic rate was generally followed by a second, which reached its summit about forty-five minutes later. Smoking caused an increase in heart rate in 72% of the subjects, a decrease in 26% and no change in 2%. After fifteen minutes the heart rate became slower than normal. The respiratory rate decreased immediately after smoking, returning to normal in forty-five minutes. Smoking caused an immediate reduction in the oxygen pulse (millilitres of oxygen used per heart beat) followed by an increase for at least forty-five minutes. The greatest physiological effects were shown by confirmed smokers who inhaled the smoke, and by persons who were unaccustomed to smoking. Women also showed more pronounced changes than men. Greater changes were produced with persons in a basal metabolic condition than by persons not in a basal condition.

An Epidemic of Brucellosis.

I. F. HUDDLESON AND MYRTLE MUNGER (*American Journal of Public Health*, August, 1940) state that for three months at Michigan State College 45 cases of brucellosis occurred with one death in the bacteriological department. Forty-one of these affected persons were college students. In addition, tests revealed that 49 others possibly had subclinical infections. Cultures of *Brucella melitensis* were obtained in 36 of the 41 cases and in two of the subclinical group, and no other species of brucella were found. Milk appears to be eliminated. Back siphonage of water from the wash-room sink into the main water line was shown to be a possibility, and curiously, a plumber who installed an autoclave in the laboratory was a victim. The evidence suggests a carrier or carriers and mass infection by association chiefly in class. Positive blood cultures were obtained from three persons who were quite free from symptoms at any time before or after culturing—active infection and clinical disease are not synonymous.

Services for Crippled Children under the Social Security Act.

R. C. HOOD (*American Journal of Public Health*, August, 1940) writes on the services for crippled children under the *Social Security Act*. He remarks that all States and territories have programmes in operation, and most health departments have a division for crippled children. The Federal grant must be matched by an equal amount from the State, and the total Congress appropriation is $2,850,000. The number of crippled children, the cost of care and the special State difficulties are also considered. An additional million dollars is devoted directly to special needs, $50,000 to poliomyelitis and $200,000 for children with heart disease. At the end of 1939, 224,000 crippled children were on State registers. It is estimated that 75% of the total do not need hospital treatment or operation. Field services by public health nurses, medical social workers and physical therapy technicians are provided for in this group. Post-graduate courses for local practising physicians in early diagnosis, prevention and treatment of crippling have been an important feature. Both extensively and intensively the States are broadening the scope of the services and the quality of care that children receive.

Importance of the Tabulation of Multiple Causes of Death.

T. A. JANSSEN (*American Journal of Public Health*, August, 1940) writes on the importance of the tabulation of multiple causes of death. He states that a study of death certificates in America shows considerable geographical variation in the frequency with which multiple causes are mentioned. The system of international classification gives preference of allotment to certain main causes. This may give little idea of the frequency of certain pathological conditions, as they are so often complications of other diseases, for example, alcoholism, acute endocarditis, acute myocarditis, coronary disease, bronchopneumonia, lobar pneumonia, acute nephritis, arteriosclerosis. Again, 10·9% of certificates involving the child-bearing state were not tabulated as indicating maternal deaths, but 50% of the deaths were charged to pneumonia, homicide, tuberculosis, and the other 50% to various causes. About 11·5% of the certificates recording septic abortion were otherwise tabulated. Similarly, 44·5% of the certificates noting "abortion non-septic" were not tabulated under this heading. Altogether 24% of all abortions as noted on certificates were tabulated under other headings. Although the official system with use of the International List of Causes of Death seeks to obtain true international comparability, the varying habits of physicians in reporting causes, the different questions on the certificate, and the varying relationship to the coroner *et cetera* interfere. The chief difficulty as tested on 1,032 identical cases referred to 18 different countries is in the methods used in tabulation, and especially in regard to multiple causation. The author makes a strong plea both for the recording of multiple causes on death certificates and for statistical analysis to show the full effect of these joint causes of death.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XXXI.

CERTIFICATION OF PATIENTS.

O, let me not be mad, not mad, sweet heaven!
Keep me in temper: I would not be mad!

THE best practical definition of insanity is that given by the late Dr. Mercier: "Insanity is disorder of conduct"; and the question to be asked by every practitioner when he is confronted by a man who is suspected of being insane is: "Does this man's conduct differ, and in what way, from that of a normal man of the same age, rank and education in similar circumstances?"

By conduct is meant everything a man says or does or—and this is equally important—what he refrains from saying or doing. Negative conduct may be insane. Every normal man goes about his business, or, if he is out of work, takes an interest in something. A man who lies in his room all day and refuses to come out for meals without reasonable cause may be insane, showing this by not doing the normal daily actions of the sane. We must not only leave undone those things we ought not to have done, but we must also do those things we ought to do, or there is no mental health in us.

It is rare for any one particular action to be of such a nature that it can be classed as insane. Smashing a shop window may be an insane act or may be the work of a smash-and-grab thief. If a citizen howls at the top of his voice in a busy city street one may have grave doubts about his sanity; but if this citizen is aged three years and has lost his mother, his conduct is exactly what one should expect. It is rather the conduct of a patient over a considerable period that has to be considered, and this has to be ascertained by personal examination of the patient and inquiries from relatives or friends.

Indications for Certification.

The following are indications for certification: (i) when the patient is in need of treatment and is unable for financial or other reasons to be treated in his own home; (ii) when he is likely to be dangerous to himself or others; (iii) when he is a nuisance to the general public; (iv) when he is wandering about with no friends able or willing to look after him. Insanity in itself is not necessarily an indication that a patient must be sent to a receiving house or mental hospital. According to the *Lunacy Act*, a patient is sent to receive care and treatment, and if he can receive adequate treatment at home there is no reason why he should be sent away.

It is forbidden for medical men to keep a private mental hospital unless this hospital is licensed by the Government and submits to various regulations, which differ in different States.

Admission of Patients to Mental Hospitals.

The regulations as to the admission of patients into mental hospitals differ in different States and are continually undergoing modification and revision, and it is advisable for a medical practitioner to make himself conversant with the regulations or, in case of doubt, to ask for a decision from the inspector-general of mental hospitals in his State. In most States a copy of the *Lunacy Act* can be bought for a very moderate price.

Voluntary Boarders.

By the voluntary boarder system any patient who understands what he is doing can ask to be taken into a receiving house or mental hospital, can be admitted if thought suitable, but has to give three days' notice (in New South Wales seven days, in Western Australia twenty-four hours) to the superintendent if he desires to leave. This notice is to give the superintendent time to communicate with the patient's friends, so as to take the necessary precautions to ensure his safety if such action is desirable.

Any physician who desires a patient to be voluntarily admitted to a mental hospital need only give the patient a letter to that effect. It is helpful to give a short statement about his mental condition, his previous personal and family history, and such details as may throw light on his disease. The best types of patients as voluntary boarders are those suffering from psychoneuroses, alcoholism and melancholia.

No voluntary boarders are taken in Tasmania or Queensland, but arrangements are being made to amend the acts in those States. In Western Australia application must be made before two justices of the peace.

Certification.

The majority of patients are admitted to a receiving house or mental hospital on the certificates of two medical practitioners. A doctor certifying must not be a relative of the patient, nor a relative, partner or assistant of the practitioner signing the other certificate. The object of this provision is to ensure that there shall not be collaboration between the relatives and doctors to detain a patient illegally. The regulation that partners, or a practitioner and his assistant, shall not certify a patient is of the greatest importance. The superintendent of the hospital to which the patient has been sent has his suspicions aroused if relatives sign the two certificates; but in the case of partners there is usually nothing to indicate this fact, and a patient may be illegally detained, in which case the penalty would fall on the partners who gave the certificates and not on the superintendent of the hospital, who had presumably acted in good faith and with reasonable care.

When a physician is actually certifying a patient it is advisable to keep in mind that the certificate is a legal and not a medical document. Its object is to declare that a certain person should have his liberty curtailed in order that he may be given care and treatment or in order that he shall not be a danger to the general public. This document must be of such a nature that a lay judge and jury will be satisfied that, on the date specified, drastic action had to be taken. Now the general public, as represented by the jury, is very suspicious of any attempts to curtail its liberty, and each juryman has the uneasy feeling that he may be illegally detained in a lunatic asylum. His prejudices are therefore against the medical practitioner who certificates the patient and the superintendent who detains him.

The following points should be carefully observed when certificates are being filled in. (1) Be sure to see and examine the patient on the date mentioned in the certificate. (ii) See the patient separately from any other practitioner. (iii) When filling in the space headed "Facts observed by myself", see that they are facts. "She sees ghosts" is not a fact observed. "She says that she sees ghosts" is a fact observed. She admits that she attempted suicide, she is restless, she refuses to enter into an ordinary conversation, are all facts that can be observed. Remember that negative facts are important. The normal man will discuss his complaints, the football matches, or anything in which he is interested, and if he refuses to do so, this is evidence of abnormal conduct. If a patient expresses delusions or hallucinations, always put these in, as they appear to have particular weight with the legal fraternity. Do not put in irrelevant facts like "his mother died in an asylum"; it may be of great importance from the point of view of a medical history, but is not a fact observed by the practitioner. (iv) In the space following "Facts communicated by others", always give the full name and address of the informant. Do not say: "His brother informs me"; the patient may have several brothers, and each will deny that he ever said anything. Facts communicated by others are not essential, but facts observed by myself are, or the certificate is illegal. (v) It is advisable to see that the patient's name is correctly spelled, his occupation is inserted, and all erasures or alterations are initialled. These certificates are valid for seven days only (fourteen in Queensland, twenty-eight in New South Wales, twenty-one in Western Australia), and it is advisable to warn the relatives of this fact; otherwise they will keep them for six months, and when the patient is refused admission to a mental hospital, will say: "The doctor should have warned us they would only last a week."

If the practitioner sees the patient for the first time, he sometimes has difficulty in knowing how to approach him. I suggest that he first obtains the history of the patient's illness and an account of his peculiar conduct from the relatives, then goes to the patient and announces that he is a doctor who has been told that the patient is ill, listens to his statements and notices his conduct. The physician can start with a physical examination (which must never be omitted) if he so desires; and if the patient refuses to allow this, a note should be made of the fact and the reasons for the patient's objection. Later, the physician can say: "Your wife has informed me that . . .", and the patient can then explain his conduct. He can be asked: "Do you hear voices when no one is present, do you see peculiar sights, do you think anyone is trying to injure you?" Hallucinations or delusions will thus be elicited.

Memory can be tested by asking the names of the principal streets of the town and what streets cross it; orientation

by asking the day and date; and general intelligence by discussing any subject of interest in the daily papers.

In the event of a medical practitioner's being called to see a patient who is violent or is likely to injure himself or others, it is advisable to call another practitioner at once, and as soon as he has certified the patient, the latter can be given an injection of one-fiftieth of a grain of hyoscine hydrobromide, so that he can be conveyed to the receiving house or mental hospital.

Sometimes a doctor in a country town knows that a man is likely to be dangerous to his wife and children, or that a local inhabitant is suffering from delusions of persecution and is threatening to murder someone. In these cases there is no legal responsibility on the doctor, but he feels a moral responsibility, and it is quite possible that the friends or relatives refuse to have the man certified or to take any reasonable action. In this case the local police should be informed that in a certain place there is "a lunatic not under proper care and control", and the police have a regular procedure by which the man can be brought before a police magistrate or justice of the peace, can be examined by a medical practitioner, and forcibly taken to the receiving house for the necessary observation.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on November 28, 1940, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, PROFESSOR W. K. INGLIS, the President, in the chair.

Compound Fractures.

DR. A. R. HAMILTON discussed compound fractures. He said that there were many omissions and a lack of references in his remarks, as they were based on personal experiences and general principles. With regard to the first-aid treatment of compound fractures, the first procedure was to expose the site of fracture, dress the wound with a sterile dressing and apply a bandage, not too tightly. If the injury was in the region of a main artery and it was not possible to control the hæmorrhage by digital pressure, a tourniquet should be applied firmly enough to stop the bleeding. Not infrequently a tourniquet was applied but half-heartedly; the venous return was blocked, but the artery was not occluded, with the result that hæmorrhage was increased. No attempt should be made to replace the fractured bones. The patients' limbs should be splinted where they lay; the last axiom was too often forgotten, with disastrous results in some cases. Sedatives should be given only if it was certain that they would not mask symptoms of other injuries.

On the patient's arrival at hospital a full examination should be made so that injuries other than the obvious one would be discovered, as not infrequently less severe injuries might be overlooked, with severe late ill effects. An attempt should also be made to determine, before the patient was anæsthetized, if tendons or nerves, or both, were injured, and if so, which ones. Surgical treatment of the compound fracture should be undertaken within six hours from the receipt of the injury. If the fracture involved a major bone, the less handling the affected part received, the better; therefore, the patient should be taken to the operating theatre on his bed, anæsthetized and then moved to the operating table after his clothing had been removed and a preliminary preparation had been carried out. If the patient was elderly or suffering severely from shock, a nitrous oxide and oxygen anæsthetic was indicated; and if hæmorrhage had been severe, preparations for a blood transfusion should be made. The skin about the wound should be shaved and prepared with ether soap, ether and iodine. If the skin edges were ragged and infiltrated with dirt, up to one-quarter of them should be excised, together with underlying fat, deep fascia and any torn or bruised muscle fibres. Loose fragments of bone should be removed, and any spikes of bone which might have penetrated the skin should be snipped off. Whilst these procedures were being carried out, frequent and thorough scrubbings with neat peroxide of hydrogen on gauze swabs cleansed the tissues most effectively, and this substance was preferable to the coagulating disinfectants, such as spirit or iodine. Any damaged large vessels should be firmly tied off, peripherally severed nerves should be sutured, and divided tendons should be lightly anastomosed in order to prevent retraction. The wound should be rendered as dry as possible, the raw surfaces should be covered with from five to fifteen grammes of sulphanilamide crystals, the fracture should be reduced with the aid of the X-ray screen if possible, and whilst the assistant maintained the limb in position the skin edges should be brought together with interrupted silkworm gut sutures, an opening being left at the optimum site for the insertion of a "Vaseline" gauze drain. The surface should be covered with "Vaseline" gauze and a thin pad, and then plaster of Paris should be applied over sterile wadding or the selected splint according to requirements. One thousand units of tetanus antiserum should be administered, and this dose should be repeated or a larger one given a week later. If operation was not undertaken within six hours of injury the case had to be treated on its merits; as a rule it was sufficient to remove foreign and loose bodies, lightly swab the wound with peroxide of hydrogen, pack with "Vaseline" gauze, reduce the fracture and put on a splint.

For the first few days after injury a temperature of 99° or 100° F. with a corresponding pulse rate was to be expected; but should the temperature jump to 103° or 104° F., the gas-forming organisms should be remembered. An X-ray examination for evidence of gas bubbles should be made, a specimen of material should be obtained from the wound for pathological examination, and if there was any evidence of gas gangrene, sulphanilamide should be given and arrangements made for immediate deep X-ray therapy.

Dr. Hamilton drew attention to the present tendency to regard plaster of Paris as the ideal medium to fix and protect compound fractures. He urged those present not to allow this tendency to override their better judgement. The object of the plaster cast was to immobilize the fracture and afford a covering to the wound, so that frequent interference was prevented. In many cases immobilization was better obtained by other forms of splinting, and by changing an outer dressing interference with the wound could be avoided indefinitely with, very often, a less disturbing odour in the ward. Skeletal traction was but rarely required in the treatment of compound fractures, and should be avoided if possible, as too often it resulted in stiff joints and osteomyelitis. Dr. Hamilton preferred to sacrifice a little length rather than risk the dangers of skeletal traction, and he reserved the latter method for the exceptional case. Such were the general principles governing the treatment; a few words on some of the individual fractures would not go amiss.

Individual Fractures.

Dr. Hamilton then discussed certain fractures individually.

Toes.—Compound fractures of toes frequently called for partial or complete amputation; other fractures were treated on their merits. A splint made of rolled zinc oxide strapping often sufficed.

Metatarsals.—Discussing compound fractures of metatarsals, Dr. Hamilton said that the foot should be fixed with a plaster of Paris shoe moulded well into the arches of the foot and extending to the tips of the toes, the latter being extended. Extension on one or more toes might be necessary in the case of oblique fractures.

Tarsal Bones.—A well-moulded plaster boot with the foot in its mid-position afforded the best fixation in compound fractures of tarsal bones.

Tibia and Fibula.—In compound fractures of the tibia and fibula the limb was best splinted by means of a plaster of Paris cast extending from the toes to half-way along the thigh, the knee being semi-flexed. Windows for dressings should not be cut.

Compound Fractures Involving the Knee Joint.—In compound fractures involving the knee joint a Thomas's splint with fixed extension should be used; an aluminium splint should be placed posteriorly and a pad of wool behind the knee.

Patella.—If the patella was severely comminuted, all fragments should be removed, the defects in the tendon should be repaired, the joint should be drained by means of a "Vaseline" gauze wick, and the limb should be immobilized on a Thomas's splint. Experience would probably prove that all compound fractures of the patella were best treated by complete excision of the bone.

Femur.—If the shaft of the femur was fractured, the limb should be placed in a Thomas's splint with back and side splints and correctly placed pads, fixed extension and a foot piece being used. A plaster splint allowed angulation of the fragments and shortening to take place. If the neck of the femur was shortened, the limb should be fixed in a Whitman spica or on a Jones's frame, according to the amount of destruction present.

Pelvis.—If the pelvis was fractured, the pelvic girdle should be encircled with zinc oxide strapping if possible; otherwise a many-tailed bandage should be used and the patient should be nursed on a Jones's frame. This permitted easy moving, averted pain and shock, and allowed constant observation of the abdomen for complications.

Spine.—A patient who had sustained a compound fracture of the spine was probably best treated in a well-padded bi-valved plaster cast from the vertex to the soles of the feet; this allowed him to be turned readily, and a constant watch to be kept for pressure or trophic sores and other complications.

Clavicle.—A fractured clavicle should be immobilized with a figure-of-eight bandage or an anterior plaster slab. A Thomas's spinal brace with a broad upper end afforded good fixation and was especially useful if much bone was missing.

Shoulder Joint.—A fractured shoulder joint should be treated in abduction on an aeroplane splint or in a plaster splint in the majority of cases, care being taken that the elbow was anterior to the frontal plane. Once the acute stage had subsided an aeroplane splint with a hinged elbow might be considered, so that elbow movements could be performed at an early date.

Humerus.—If the shaft of the humerus was fractured, the arm should be placed at the side, the wrist slung to the neck with a cuff and collar, and suitably twisted aluminium splints applied to the outer and inner sides and the back of the arm, one of them extending over the shoulder for fixation purposes. A plaster mould could be applied to the same aspects, care being taken to renew it at frequent intervals as the swelling subsided. The humerus was a difficult bone to immobilize, owing to the mobility of the shoulder girdle. It might be necessary to apply extension to the fracture; if so, a Jones's and not a Thomas's arm splint should be used. It was inadvisable to persevere with extension for long periods without X-ray examinations, as too much lengthening was worse than too little. The former almost invariably led to non-union. If the neck of the humerus was fractured, the treatment was the same as for the shoulder joint.

Elbow Joint.—The majority of elbow joint fractures could be treated by cuff and collar fixation alone, the dressing being held in place by a loosely applied bandage. If much comminution was present, a plaster mould along the back and two sides of the forearm assisted in maintaining position. Early active movements were called for. If ankylosis appeared to be inevitable, the elbow should be placed in the optimum position for the person concerned, and fixed with either a cuff and collar if flexion was desired, or a plaster mould if partial extension was aimed at.

Forearm.—If both bones of the forearm were fractured, owing to the risk of cross-union the arm should not be immobilized in full supination. Immobilization could be effected either by a plaster splint from the palm of the hand to a few inches above the elbow, the latter being placed at a right angle, or by suitable aluminium splints, made in the form of a short cock-up for the hand and bent at a right angle at the elbow. If only one bone was fractured, suitably twisted aluminium splints could often be used, although fixation in a plaster splint was to be preferred if there was a tendency for fragments to override or if much comminution had occurred.

Wrist.—A fractured wrist should be immobilized with a Crabbe's splint or with a plaster splint from just below the elbow to a point distal to the crease of the palm; the hand must be dorsiflexed.

Metacarpals.—If the metacarpals were fractured, a long aluminium or cock-up splint with wire extensions attached might be necessary in the more severe injuries, extension being made by means of tape fastened to the finger with collodion flexile. A compound fracture of the neck of the metacarpal often presented a difficulty, in that this fracture was best treated with the finger fully flexed; yet this form of treatment in compound injuries invited a finger stiff in flexion. It was better to sacrifice alignment to some extent, if that was necessary, to allow a finger to maintain sufficient extension. The hand should never be treated on a straight splint; this applied to fingers as well as to metacarpals, as the resulting stiffness was often irremediable.

Fingers.—In compound fractures of fingers it was important to obtain an anatomical reduction of the fracture and to splint the finger on a Glissan splint. Movements should be allowed as soon as possible. Compound comminuted fractures of terminal phalanges were often best treated by amputation of most of the phalanx, together with the nail and its matrix.

Thumb.—A fractured thumb should be splinted in abduction with extension if necessary.

DR. GEORGE BELL congratulated Dr. Hamilton on his paper. He said that it was to be regretted that so few members were present; the paper would have been of value particularly to resident medical officers and casualty surgeons. Dr. Bell supported what Dr. Hamilton had said about the need for proper first-aid treatment. During the war of 1914-1918 appropriate first-aid treatment of compound fractures of the femur made a great difference to the immediate and end results. The most important feature was the application of a Thomas's knee splint soon after the injury and as near the place where the casualty occurred as was possible. The late Mr. Maynard Smith had been much impressed by the excellent condition of a man who had sustained a compound fracture of the femur and was leaving a casualty clearing station with the limb in a Thomas's splint. Mr. Smith, who had had knowledge of the results of treatment before the Thomas's splint had come into general use, found it difficult to believe that the man really had a compound fracture of the femur, his condition was so good. Dr. Bell then said that the French during the last war had used ether for cleansing wounds associated with fractures. Dr. Bell had used ether for years, and also peroxide of hydrogen.

Dr. Bell considered that judgement should be withheld in connexion with the results of injuries sustained by the men rescued from Dunkirk; those with very severe injuries would be unlikely to have reached England. The results were at present rather being judged by the cases actually dealt with in England. Dr. Bell then referred to the term *débridement*, which he considered to be somewhat like the blessed word "Mesopotamia". He thought that it might easily be replaced by the word "excision" or even "decontamination". All that it implied was the getting rid of damaged tissues by thorough excision. He urged those present to read Trueta's work. Fixation of the limb was important after the contamination of the wound had been dealt with. The main virtue of plaster of Paris was that it eliminated meddlesome dressings and gave rest to the wounded limb. The first essential was excision of the wound. This was not always easy. It was hard to be sure that every fissure and crevice had been cleaned out. With certain wounds, in close contact with main blood vessels or nerves, it was necessary to take a chance, and thorough excision was impossible. Dr. Bell agreed with Dr. Hamilton that the Thomas's splint was excellent for fractures in the region of the knee joint, especially when a posterior splint of aluminium or some such material was applied to give rigidity. Dr. Bell thought it wise to give a second dose of tetanus antiserum. During the last war the orders were to give three doses. Dr. Bell then referred to the question whether sulphanilamide should be given prophylactically in bad compound fractures. He thought that it should be given, and in full dosage. He understood that there had been some objection to the use of sulphanilamide in the presence of shock. The prognosis in compound fractures depended a great deal on the amount of contamination of the wound. Even the locality where the wound occurred might have an influence on the amount of infection. He had been informed by a medical officer that on the Western Front in the region of La Bassée, infection of wounds was very severe. When the fighting became open the wounds sustained were quite different. There were more bullet wounds and they were much cleaner. During the present war explosives were more powerful and there was more pulping of the tissues, and the fragments of high explosive shells were often much smaller. With regard to gas gangrene, Dr. Bell said that there seemed to be something in treatment with X rays; but it was necessary to be careful and to watch that the treatment was not persisted in too long. In Dr. Bell's experience, in all cases in which gas gangrene extended throughout the whole length of the thigh, the patient died. There were cases in which the wound became very rapidly gangrenous as a result of infection by microorganisms. He recalled one case in which the whole limb became black in forty-eight hours; but at that period no facilities were available for finding out which organism was responsible. Dr. Hamilton had mentioned the taking of a swabbing if there was any suspicion that the wound was becoming infected. Dr. Bell thought this a wise procedure. In a fairly slack interval in the winter at one casualty clearing station they took swabbings from wounds at the time of operation, and if *Bacillus perfringens* was found the patient was very carefully watched for signs of gas infection.

Dr. Bell went on to say that the treatment outlined by Dr. Hamilton was excellent. Dr. Bell could remember at Sydney Hospital only one case that had really beaten him. The patient was a man with a compound fracture of the lower end of the tibia, and he had a very dirty foot. The wound was thoroughly cleansed, but it suppurated, and an amputation had to be performed. Later it was found that the patient was a tertiary syphilitic. This was the only case that had gone wrong in Dr. Bell's experience when this form of treatment was used. It was worth while spending time cleansing the wound thoroughly. Only recently a naval rating, while playing football, had sus-

tained a compound subastragaloid dislocation and was taken immediately to the Prince of Wales Hospital with the bone protruding. After decontamination the limb was immobilized in plaster of Paris and a perfect result was obtained. Dr. Bell remarked that this case illustrated the great importance of early treatment of compound fractures. Students should be taught that they were just as "urgent" as ruptured gastric or duodenal ulcers.

COLONEL R. A. MONEY congratulated Dr. Hamilton on the clear way in which he had expressed his opinions. Colonel Money once more stressed the need for proper first-aid treatment of compound fractures. He said that the Civil Ambulance officers did not seem to realize the importance of the Thomas's splint. Colonel Money had seen patients with fractures of the femur arrive at the Royal North Shore Hospital in a Thomas's splint; the ambulance officers took them to the X-ray room in a splint, and then took off the splint because it was theirs and they had to take it away. The patient was then "dumped" here, and had to be lifted two or three times without the splint. Colonel Money wondered whether this point was sufficiently stressed in first-aid treatment. During the preceding few days he had read an article from the Mayo Clinic on the subject of treating compound fractures. It was easy to be certain of what to do when the patient was treated within six hours of the injury; but in the treatment of patients whose injuries had been sustained more than six hours previously, or when infection had already set in, a new method had been adopted at the Mayo Clinic. It was a cross between the Trueta treatment and the Carrel-Dakin method. After *débridement* the wound was washed out with peroxide of hydrogen and then tubes were put in, and the wound was irrigated three times a day with a saturated solution of sulphanilamide. At the Mayo Clinic they were also keen on giving sulphanilamide by mouth, not only prophylactically, but also therapeutically. Colonel Money then referred to the present procedure by which the troops were all given two doses of tetanus toxoid, which was supposed to confer lasting immunity. He wondered whether it was important to preserve exactly the interval of six weeks between doses. He understood that this time interval was very important; but was it? If the procedure was comparable with the administration of diphtheria toxoid, the time interval was very important in building up lasting immunity.

DR. T. W. LIPSCOMB asked Dr. Hamilton to speak further on drainage with "Vaseline" gauze. He said that from a surgical point of view no gauze was a drain except only while it was dry; once it was wet it became a plug. Referring to the question of injections of tetanus toxoid, Dr. Lipscomb said that he knew that the procedure in English military camps was to give a second injection between five and six weeks after the first, according to convenience. There were no reactions, and the dose was one cubic centimetre of toxoid. The interval was not less than five weeks and not more than six. On the question of the men from Dunkirk, Dr. Lipscomb said that ultimately 1,250 had been admitted to Saint Mary's Hospital. They were some days in arriving there, and the men with the worst injuries were not taken further than hospitals at the seaside. At Saint Mary's Hospital in only one case did a gas gangrene infection lead to amputation. The Ministry of Health had laid it down that treatment was to be by sulphanilamide, excision and the use of serum, but if reasonably possible amputation was not to be performed. At one hospital to which these men were taken on the coast there were a very large number of amputations. An official from the Ministry of Health went to the hospital to see what was happening, and he had to admit that the injuries were dreadful. The absence or lessening of incidence of gas gangrene as compared with the last war was attributed to the fact that they were fighting in summer time, so that there was no mud; and the mechanization of the forces meant that there were fewer horses and less manure.

DR. H. A. SWEETAPPLE also thanked Dr. Hamilton for his paper. He regretted that only a dozen people had thought it necessary to attend, especially as the paper was on a very live topic. Dr. Sweetapple agreed with everything Dr. Hamilton had said. Merely by way of comment, he thought that treatment might be classified under four headings: (i) promptness, (ii) thorough *débridement*, (iii) avoidance of meddlesome dressings, and (iv) complete and absolute and uninterrupted immobilization. One thing about *débridement* was to avoid, if possible, the use of catgut. It was stupid to remove dead tissue and replace it by other dead tissue, which was all that catgut was. It was surprising how few vessels needed to be tied off; sometimes none needed it. With regard to promptness, it was necessary to bear in mind that the patient was probably suffering from shock, and the six-hour interval might have to be exceeded if treatment for shock was to be continued for a longer period.

DR. A. T. ROBERTS also thanked Dr. Hamilton for his paper. With regard to the first-aid treatment of compound fractures, Dr. Roberts said that at Newcastle any ambulance difficulty appeared to have been overcome. The ambulance officers always applied a Thomas's splint in fractures of the leg, and the fractured limb remained in the splint when the patient arrived at the hospital. An exchange of splints between the hospital and the ambulance had been arranged, and the system worked well. On one point Dr. Roberts dissented from the views of Dr. Hamilton; that was the question of *débridement*. At Newcastle they had worked out a system of treatment with which they had obtained very satisfactory results. Their procedure was not to worry much about *débridement*. The main object of the primary treatment of compound fractures was to get the patient fixed up as quickly as possible on account of the shock and to stop the hæmorrhage. Ineffectual hæmostasis caused a rise of temperature later. The more *débridement* and the more cutting of tissues that were done, the more oozing and bleeding occurred. They trimmed away damaged tissue as far as possible, but they did not worry about complete *débridement*, and their results were very satisfactory. With regard to Dr. Lipscomb's remarks about "Vaseline" gauze as a plug, Dr. Roberts said that one thing that "Vaseline" gauze was not was a plug. It did not stop bleeding; the blood went on oozing out between the skin and the drain. "Vaseline" gauze was not a plug, but a loose floppy drain. In most instances they did not use gauze; they filled the wound with "Vaseline" and put a slab of "Vaseline" over the wound and left it alone.

DR. H. H. JAMIESON asked whether in the presence of a compound fracture of the leg it was considered to be a perfectly safe procedure to fix the leg in plaster straight away. He had done this in two cases in which the leg swelled considerably in the following twenty-four to forty-eight hours, the plaster had to be cut off and gas gangrene developed. Dr. Jamieson felt that it was not a perfectly safe procedure and that in some cases there was a certain risk of interference with the circulation if the limb was fixed in plaster before the swelling went down.

Dr. Roberts asked Dr. Hamilton whether he thought that the use of plaster of Paris on these fractured limbs was more of an inducement to the development of gas gangrene than other open methods of treatment.

Dr. Hamilton, in reply to Dr. Bell, who had introduced the question of using ether instead of hydrogen peroxide for washing out fracture wounds, said that he preferred hydrogen peroxide because its foam exerted a mechanical action in lifting damaged tissue and dirt from the wound. Dr. Hamilton then referred to the fact that the use of plaster of Paris prevented meddlesome dressings. He said that one of the essentials for successful orthopædic work was the avoidance of such dressings. It was necessary to have in the hospitals proper teams of nurses, who could be trained along these lines. Dr. Hamilton not only thought that the Thomas's splint would be the best for many years; he thought that it would never be replaced. With regard to the use of sulphanilamide in compound fractures, Dr. Hamilton said that his experience was rather limited; but in two cases in which he had used it, the results had been very satisfactory. In reply to Dr. Lipscomb, Dr. Hamilton said that he still felt that a "Vaseline" gauze drain was a drain and not a plug. The gauze had to be well saturated with "Vaseline"; a little was of no use. In reply to Dr. Sweetapple, who had raised the question of the use of catgut sutures, Dr. Hamilton said that most vessels would cease bleeding if they were merely twisted with artery forceps. Sutures were needed only for larger vessels. No trouble from shock within the six-hour period had been experienced when gas anæsthesia was used and "Neosynephrin hydrochloride" was administered. Dr. Hamilton said that extensive *débridement* was to be used only when the wound was very ragged and infiltrated with dirt; otherwise it was kept to a minimum. Dr. Hamilton agreed that it was important to stop hæmorrhage; that was a point made by Thomas in 1875. He had laid stress on the use of hydrogen peroxide. In reply to Dr. Jamieson, Dr. Hamilton said that he had only once had to split the plaster cast on account of swelling of the tissues; he had never had gas gangrene occur in the circumstances mentioned by Dr. Jamieson. Dr. Hamilton wondered whether Dr. Jamieson padded the plaster splint.

Dr. Jamieson said that he used a fine layer of cotton wool as padding.

PROFESSOR INGLIS, from the chair, on behalf of the New South Wales Branch of the British Medical Association, thanked Dr. Hamilton for his paper, which had provoked a practical and interesting discussion.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 7, of January 16, 1941.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Termination of Appointment.—The appointment of Surgeon Lieutenant (for Short Service) Walter Mario Butler is terminated, dated 16th December, 1940.

Extension of Appointment.—The appointment of Surgeon Captain William James Carr, C.B.E., is extended for a period of one year from 30th January, 1941.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointments.—Gordon David Ley is appointed Surgeon Lieutenant, dated 1st December, 1940; Surgeon Commander Wilfrid John Robert Nickson, Retired List, is appointed Port Division Naval Medical Officer, Newcastle, dated 3rd September, 1939.

AUSTRALIAN MILITARY FORCES.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captain.—James Ronald Turner, 6th December, 1940.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps.

To be Captains (provisionally).—Walter Edward Fisher, 31st October, 1940; Howard Bullock, 8th November, 1940; Norman Duffin Barr, William Clover Howie Epps and Edward Morell Cortis, 26th November, 1940.

Honorary Captain N. Rau is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 19th June, 1940.

The provisional appointment of Captain N. M. Stewart is confirmed.

Captain A. H. Gee ceases to be seconded, 20th September, 1940.

SOUTHERN COMMAND.

Third Military District.

Australian Army Medical Corps.

To be Captains (provisionally).—Robert Wall and Douglas Robert Leslie, 31st October, 1940.

Honorary Captain G. Forsyth is appointed from the Reserve of Officers (A.A.M.C.), and to be Captain (provisionally), 18th November, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captain.—William Reade Lane, 7th December, 1940.

The resignation of Honorary Captain J. J. Laing of his commission is accepted, 18th November, 1940.

Fourth Military District.

Australian Army Medical Corps Reserve.

To be Honorary Captain.—Ernest Stirling Chapman, 12th November, 1940.

Sixth Military District.

Australian Army Medical Corps.

To be Major (temporarily).—Captain W. J. Freeman, 25th September, 1940.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The following temporary Squadron Leaders are granted the temporary rank of Wing Commander with pay of that rank with effect from 1st January, 1941: (Acting Wing Commander) W. D. L. Farrar and H. J. Melville.

The following Flight Lieutenants are granted the temporary rank of Squadron Leader with pay of that rank with effect from 1st January, 1941: S. W. Lush, A. A. Green, J. G. Lauder and P. J. Egan.

Citizen Air Force: Medical Branch.

Temporary Squadron Leader P. J. Benjamin is granted the temporary rank of Wing Commander with pay of that rank with effect from 1st January, 1941.

The following Flight Lieutenants are granted the temporary rank of Squadron Leader with pay of that rank with effect from 1st January, 1941: V. E. Knight, J. C. Laver, F. F. Ellis, G. M. Oxer, I. R. Horn, T. Burfitt, C. J. Cummins, D. J. Prentice, M. L. Creightmore, A. H. Penington, C. P. Ley, I. H. Cuming, (Acting Squadron Leader) J. C. Fulton, J. M. Rainbow, (Acting Squadron Leader) P. R. Delamothe, S. B. Forgan, J. D. Russell, J. L. R. Carter, C. P. Hudson, H. D. Phipps, (Acting Squadron Leader) J. J. W. Flynn and (Acting Squadron Leader) T. A'B. Travers.

The probationary appointments of the following Flight Lieutenants are confirmed: M. H. B. Robinson, B. J. Mulvany, H. A. F. Rofe, J. A. Bond, R. V. Pridmore, W. F. H. Crick, R. W. Hazelton, D. L. Peate, G. G. Burniston, A. J. Campbell, D. C. Howle, G. C. Wilson, J. H. Bilton, W. C. Holley, W. P. H. Dakin, A. L. Kennett, W. McL. Borland, J. J. W. Flynn, T. A'B. Travers, P. E. Breheny, D. J. Biddle, G. W. Davies, C. A. McHardy, H. C. Stone, W. L. Fowles, A. J. Terry, J. L. Walter, M. J. Barrett, N. E. Allan, A. G. Crain, M. J. Quealy, R. T. McCaul, P. A. Benbow, C. L. H. Bott, J. C. Burns, R. J. Dingle, H. R. Hall and A. F. B. Stone.

The following Flight Lieutenants are granted the acting rank of Squadron Leader with effect from 1st January, 1941: W. McL. Borland, W. M. Lemmon and A. J. Terry.—(Ex. Min. No. 8—Approved 9th January, 1941.)

Flight Lieutenant G. E. Phillips is transferred from the Reserve to the Active List, and is granted the temporary rank of Squadron Leader with effect from 17th December, 1940.

Flight Lieutenant S. F. McR. Yeates is transferred from the Reserve to the Active List with effect from 16th December, 1940.

The following Flight Lieutenants are transferred from the Reserve to the Active List, with effect from the dates indicated: A. W. Bayley, 2nd December, 1940; and A. J. W. Ahern, 3rd December, 1940.

Alexander Jarvie Hood Stobo, M.B., Ch.M., M.R.C.P., F.R.A.C.P., is granted a commission with the rank of Flight Lieutenant and the temporary rank of Squadron Leader, with effect from 1st December, 1940.

John Kenneth Gabriel, M.B., B.S., D.G.O., is granted a commission on probation with the rank of Flight Lieutenant, with effect from 9th December, 1940.

The following Flight Lieutenants are transferred from the Reserve to the Active List, with effect from 9th December, 1940: C. H. Anderson, J. Catarinich, K. S. Harrison, O. W. Leitch, N. L. Newman and H. M. Webber.

Reserve: Medical Branch.

Hubert Clement Finn, M.B., Ch.M., is granted a commission on probation with the rank of Flight Lieutenant with effect from 16th December, 1940.—(Ex. Min. No. 6—Approved 14th January, 1940.)

Flight Lieutenant J. Russell relinquishes his commission, with effect from 18th November, 1940.—(Ex. Min. No. 2—Approved 9th January, 1941.)

Flight Lieutenant W. W. Ingram relinquishes his commission with effect from 1st December, 1940.—(Ex. Min. No. 11—Approved 9th January, 1941.)

Correspondence.

A NEW DIAGNOSTIC METHOD.

SIR: In THE MEDICAL JOURNAL OF AUSTRALIA, November 16, 1940, I described a technique of electro-diagnosis which was applied to the more accurate diagnosis of visceral lesions. Since the article was written many months ago, a further observation has been made, appreciation of which will obviate a source of confusion in carrying out the test.

It is appropriate to summarize briefly the main principles entailed in the procedure before describing the complication which is of present concern.

It has been observed that the specific points to which visceral pain and tenderness are referred to on the MacKenzie principle, prove also to be electric terminals which actively absorb electrical current from a Bristow apparatus. The effect of this absorption on the patient is that the current proceeds along the somatic-visceral arc to the deep-seated lesion and excites a characteristic objective sensation in the region of the lesion. At the point of current entry there is a localized response of a clonic nature.

The added observation here tendered concerns an overflow phenomenon.

It was noted in the original article that when a hypersensitive viscero-somatic arc is aroused by a unilateral lesion

such as that of gall-bladder pathology, the electrical response is not confined to the classical area of gall-bladder tenderness in the right hypochondrium. A contralateral area in the left hypochondrium also exhibits a modified capacity for current absorption and clonic response. The association of objective pain, however, is absent on this side.

It is logical to conclude that just as pain in gall-bladder lesions may be referred to the left hypochondrium or even precordium, so, objectively, there is a nervous overflow from the right to the left sections of the segmental arc concerned. No doubt this dispersion, which has led gall-bladder lesions to be mistaken for anginal disorder, is reared on some normal mechanism of physiological adjustment which operates between the two sides of the body; and once more, pathology is reared on disturbed physiology. It is important to note that this bifurcation of response is commonly encountered in the presence of any unilateral lesion, such as in appendicitis and states of ileus. If the existence of dual points of excitation is not appreciated the investigator may be misled into suspecting the presence of two separate lesions.

When two distinct lesions do occur, the test may allow of their differentiation. Thus a middle-aged male gave a history of vague indigestion associated with upper abdominal discomfort after meals. The test not only disclosed a circumscribed area of focal sensitivity in the region of the hypochondrium and of the specific type encountered in gall-bladder lesions. A tongue of hypersensitivity which extended towards the mid-line was also disclosed. In asking for an X-ray report it was suggested that the probable lesion was gall-stones associated with periduodenal adhesions. The X-ray report states: "There is a large calculus in the fundus of the gall-bladder and, as you suspect, duodenal adhesions."

Speaking generally, the test would require less finesse in its use if visceral response were confined to states of organic disorder. This is not the case, and states of functional irritability produce reactions of a more tardy nature. Further, many new patterns of referred electrosensitivity are disclosed in the investigation of disease, a situation which is inevitable in connexion with a technique which elicits a refinement of data inaccessible to coarser methods of palpation. Finally, construction of a topographical chart which illustrates and sorts all possible reactions can only be advanced by adopting also a system of reverse observation which charts surface reactions in a series of cases in which final diagnosis has been arrived at by alternative methods.

Yours, etc.,

96, Balaclava Road, Caulfield, Victoria.
FRANK TRINCA.

Undated.

SO-CALLED REFLEX ANURIA.

SIR: I was pleased to read the letter by Dr. W. John Close in THE MEDICAL JOURNAL OF AUSTRALIA, January 18, 1941, in which he criticizes certain opinions expressed by me in my article on so-called reflex anuria, which appeared in the journal of December 7, 1940, page 602.

Dr. Close agrees that in children, such as those quoted in my article, calculi may be a frequent cause of anuria; and in the young he is open to persuasion that it may be the only cause. From his other comments I feel justified in concluding that he is in agreement about there being no renal secretory suppression in these cases. Dr. Close joins issue with me, however, where adults or adolescent cases are concerned; and holds the view that these cases may be due to spasms at the uretero-pelvic junctions, for one must postulate a bilateral lesion. I prefer to regard cases of post-operative suppression, whether in children or adults, as being due to a mechanical block, generally of the nature of calculi. One must exclude from consideration cases of true anuria, either prerenal or renal, which all agree sometimes occur in elderly people, or others with damaged kidneys. The cases which I have brought together in my article come under a different category and are not cases of anuria at all, but cases of retention of urine above an obstruction, which may be at the uretero-pelvic junction or somewhere along the course of the ureter.

Dr. Close quotes post-operative vesical retention, and also paralytic ileus as examples of neuro-muscular defects, which may follow operations, the inference being that these are parallel cases. They are, however, not comparable conditions to the cases under consideration. The emptying of the bladder can hardly be compared with the emptying of the renal pelvis; and in paralytic ileus, even if one accepts the theory of neuro-muscular defect in these cases, the defect is not confined to any particular sphincter. With regard to the cases quoted by Dr. Close in support of his theory of spasm at the uretero-pelvic junction. One was an elderly woman where suppression of urine followed an extensive operation. Dr. Close admits that there were several features in this case suggestive of a calculus. There was obstruction to the passage of a ureteral catheter, there was pain in the right iliac fossa, and a plain X ray revealed a shadow suggestive of a calculus in the lower ureter. However, no calculus was discovered at operation on one kidney. The renal pelvis contained urine under pressure, and there was obstruction at the uretero-pelvic junction. The pelvis was incised and the obstruction immediately disappeared, and it was now possible to pass a catheter along the ureter. The obstruction had been present for seven days. Dr. Close states that "it could have been due to no other cause than a neuro-muscular incoordination". To carry conviction, such a statement should be supported by indisputable evidence. It is very unlikely that the opening of the distended pelvis would suddenly and completely relax a sphincter which had been in a state of spasm for seven days. Indeed I cannot conceive of any sphincter remaining in a state of spasm for seven days. I venture to suggest that not finding a calculus in a case of this kind is not conclusive evidence of its absence, however skilful the surgeon may be. Assuming that there was a small calculus at the uretero-pelvic junction in this case, when the kidney was exposed, it might easily have been released when the pressure behind it was removed; and such a calculus would be elusive and might readily escape detection. Another case quoted by Dr. Close was an elderly patient, where suppression of urine followed cholecystectomy, which Dr. Close remarked was not a mistaken diagnosis, but does not quote any evidence of having done any urological investigation to exclude the possibility of the patient having had a stone in addition to the cholecystitis. This patient had suppression of urine for three days and was given intravenous drip therapy, together with the administration of one single dose only of eserine and papaverine; and this one injection was presumed to have completely and permanently relieved a spasm which had been present for three days. If spasm at the uretero-pelvic junction be brought forward as a cause of the retention of urine, we have to believe that the spasm may exist without intermission for periods up to a week or more, and that one injection of a drug will completely and permanently relieve it. I can understand some mechanical block, such as a calculus, being relieved by one dose of such a drug, but not a prolonged spasm alone, if such a condition were possible. Then if we are to assume that spasm is sometimes a cause of suppression of urine in these cases, the position of the spasm must vary, because in only one of my cases was the obstruction at the uretero-pelvic junction, and in the others which I had an opportunity of examining it was low down in the ureter. Hence I consider that all the evidence points to a calculus, or possibly a looser agglomeration of crystals, as being the cause of that condition which has hitherto been described as post-operative anuria. There seems to be very little doubt that the cases following the administration of sulphanilamide are due to such an occurrence. Bilateral calculi are the one well-recognized and undisputed cause of double ureteral obstruction and I fail to see why one should have to bring forward different explanations for post-operative cases and for cases occurring in children and in adults. It is interesting and significant that the explanations offered by my two critics so far are quite different, the one suggesting œdema of the ureters and the other spasm at the uretero-pelvic junction as the probable cause.

Yours, etc.,

141, Macquarie Street, Sydney.
P. L. HIPSLEY.

Undated.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Act of* 1939, of Queensland, as duly qualified medical practitioners:

Neville, Thomas Russell, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.

Muller, Edmund, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.

Reimers, Eileen Harriet, M.B., B.S., 1940 (Univ. Queensland), 17, Gladstone Road, South Brisbane.

Salter, Roger Burnett, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.

Sullivan, John Stephen, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.
Wilson, Harry Gilmore, M.B., B.S., 1940 (Univ. Queensland), General Hospital, Brisbane.

The following change of name has been registered:

Roxon, Isadore (formerly Ropschitz, Izydor), M.D. (Univ. Padua), St. Kilda Flats, Moray Street, New Farm.

Post-Graduate Work.

LIBRARY SEMINARS AND CLINICO-PATHOLOGICAL CONFERENCES.

THE New South Wales Post-Graduate Committee in Medicine announces that library seminars and clinico-pathological conferences, arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology, are held every month in the lecture hall at the Prince Henry Hospital, Little Bay, at 4.30 p.m., on the second and fourth Mondays respectively, public holidays excluded.

The next library seminar will be held on Monday, February 10, 1941, and the subjects will be: (*a*) "Pancreatic Lithiasis", (*b*) "Encephalitis".

The usual monthly clinico-pathological conference will be held at the Prince Henry Hospital on Monday, February 24, 1941, but an additional conference has been arranged to be held at Royal Prince Alfred Hospital at 4.30 p.m. on Wednesday, February 12, at which the cases for diagnosis and discussion will be: (*a*) "A Patient with Splenomegaly", (*b*) "Death in a Case of Acute Thyreotoxicosis".

An invitation to be present at the meetings is extended to all medical practitioners.

Obituary.

BASIL JOHN ADAM.

WE regret to announce the death of Dr. Basil John Adam, which occurred on December 7, 1940, at Parkville, Victoria.

ALLAN JOHN BOTHAMLEY.

WE regret to announce the death of Dr. Allan John Bothamley, which occurred on December 30, 1940, at Fitzroy, Victoria.

EDWARD AUGUSTUS SPOWERS.

WE regret to announce the death of Dr. Edward Augustus Spowers, which occurred on January 2, 1941, at East Melbourne, Victoria.

FRANCIS ALDERSLEY WOOD.

WE regret to announce the death of Dr. Francis Aldersley Wood, which occurred on January 4, 1941, at Leongatha, Victoria.

OTHO PARKES FOX.

WE regret to announce the death of Dr. Otho Parkes Fox, which occurred on January 24, at Mortdale, New South Wales.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Rubinstein, Kusiel, M.D., 1936 (Univ. Paris), Balmain and District Hospital, Balmain.

Books Received.

"Textbook of Medicine", edited by J. J. Conybeare, M.C., D.M., F.R.C.P.; Fifth Edition; 1940. Edinburgh: E. and S. Livingstone. Demy 8vo, pp. 1151, with illustrations. Price: 24s. net.

"The 1940 Year Book of Industrial and Orthopedic Surgery", edited by C. F. Painter, M.D.; 1940. Chicago: The Year Book Publishers. Crown 8vo, pp. 484, with illustrations. Price: $3.00 net.

Diary for the Month.

FEB. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 5.—Western Australian Branch, B.M.A.: Council.
FEB. 5.—Victorian Branch, B.M.A.: Branch.
FEB. 6.—South Australian Branch, B.M.A.: Council.
FEB. 7.—Queensland Branch, B.M.A.: Branch.
FEB. 11.—Tasmanian Branch, B.M.A.: Branch.
FEB. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 14.—Queensland Branch, B.M.A.: Council.
FEB. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia: all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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